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Archives of Neurology and Psychiatry

VOLUME 14 JULY, 1925 NUMBER 1

DIAGNOSIS OF SPINAL TUMORS

WITH ESPECIAL CONSIDERATION OF ROENTGEN-RAY TREATMENT OF TUMORS AND OF SYRINGOMYELIA *

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Few words need be said concerning the nosography of spinal tumors. Their classical symptoms are well known, as are also the changes in the spinal fluid. In the presence of tumors, the fluid may show a great quantity of protein, a low pressure quickly following the release of fluid, and, in cases of block, the absence of the normal movements in the fluid pressure, in respiration, heart action and the Queckenstedt test.

The cellular condition of the fluid may be said generally to be normal. It is on this point that our experience is, to a certain degree, different. For a long time, we have considered that three cells per cubic millimeter was the highest number that could be recognized as non-pathologic, but three may perhaps be regarded as being too high. We have based this opinion on general clinical experience, although this may even be regarded as an insufficient basis on which to judge such a problem. Our Danish colleague, Neel, who has made investigations on this point, has reached the conclusion that the highest limit for the number of cells should be 1 per cubic millimeter. For practical clinical purposes, we dare not follow Neel, because we must take into consideration the natural source of errors of every clinical method of investigation. From a practical standpoint, Neel's results do not appear favorable to us.

In eight cases of tumor of the meninges found in vivo or at post-mortem, three had from five to seven cells, and two had three cells. In five cases in which the diagnosis of tumor from the meninges seemed to be certain (but not confirmed by necropsy), one had from four to five cells, one from seven to eight cells, and one from thirteen to twenty-three cells. In the last case, however, there were also tumor cells in the spinal fluid. We find, then, that in about one half of the cases of tumors of the meninges, there have been an increased number of cells. Attention is called to this fact here, since this conclusion does

* Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June 1924.

not correspond to the general opinion. It must be admitted, however, that a slight rise in the number of cells in the fluid is not sufficient grounds for the diagnosis of tumor of the meninges.

The following case reports are illustrative of the benefits of roentgen-ray treatment.

REPORT OF CASES

Case 1.—A woman, aged 27, came to the clinic in June, 1921. She had had symptoms of a tumor below the right mammilla in February, 1920, and the tumor had been extirpated six months later. It was diagnosed as myxosarcoma. There was a recurrence in December, 1920, which necessitated another operation. In April, 1921, there was a second recurrence, at which time she had pain in the legs. Examination showed a certain weakness of the lower extremities, especially on the right side, but no localized paralysis. The patient was unable to stand, because movement brought about severe attacks of pain; even moving the bed covers caused pain. There was no loss of sensibility, and the reflexes were normal. Roentgen-ray examination of the spine was negative. The spinal fluid at the first lumbar puncture was amber color. The Nonne test was strongly positive. There were four cells per cubic millimeter, and the Wassermann test was negative. The diagnosis of a diffuse myxosarcoma extending along the spinal cord was made. Evidently, it had not produced any distinct compression of the cord, and the symptoms indicated an irritation of the spinal meninges.

Roentgen-ray treatment was given, with very good results. The patient showed considerable improvement, the attacks of pain disappearing with the first treatment. The ability to walk returned, and after the third treatment she was able to pick up things from the ground. There were never any particular symptoms showing marked loss of function. At this time, the left foot was slightly weaker than the right. Various roentgen-ray examinations of the spine were always negative. Lumbar puncture in September, 1921, showed seven cells; in June, 1922, three cells; and in August, 1922, two or three cells. The Nonne test was positive, but the color of the fluid was, later, never increased. We were informed by letter that she developed a metastasis in the left groin in November, 1922, and later a generalized metastasis with ulceration and sepsis. She died in March, 1923.

Case 2.—A patient, aged 40, came to the clinic on July 28, 1921, with a history of pain in the back and spine, which came on suddenly in February, 1920. The pains were especially severe at night, but were not accompanied by other symptoms. She continued to perform her household duties. In November, 1920, she was compelled to sit up at night because of the severity of the pains, which at that time radiated around the chest, especially on the right side. Weakness of the lower extremities developed in March, 1921, and gradually the gait became more disturbed. Examination revealed a spastic paresis of the lower extremities, more pronounced on the right side. The patient was able to sit up in bed without using her arms, although this was difficult. There was a bilateral Babinski sign; the abdominal reflexes were lost, but there was no loss of tactile sensation. Sensation for pain was somewhat impaired in the region below the mammillary plane; deep sensibility was impaired in the toes. There was a positive Romberg sign, and the gait showed paresis.

Lumbar puncture revealed normal pressure (120). The fluid was yellow, and coagulated spontaneously within a short time, precluding the possibility of

determining the number of cells present. The Wassermann test was negative in the blood and spinal fluid. Roentgen-ray examination showed that the intervertebral disk between the third and fourth dorsal vertebrae was narrow and deformed. There was also alteration in the disk between the fourth and fifth dorsal vertebrae. In August, deep sensibility was more disturbed, and there was difficulty in sitting up. On the right side, at the umbilical plane, there was an area of impaired tactile sense. The paralysis of the lower extremities gradually progressed until the end of August, at which time there was paralysis of the right side. Later, the appreciation of movement in the feet was entirely lost. Sensation for pain was impaired greatly below a level of about 10 cm. below the mammillary line, but tactile sense was normal.

The spinal fluid findings indicated that a tumor was present, and the history of the case coincided with this diagnosis. In the beginning, however, there was no sensory disturbance to aid in localizing the tumor, but later when sensory disturbances appeared, there was sufficient evidence of a localized lesion. Laminectomy at the level of the fifth and sixth dorsal vertebrae was proposed, and on August 30, the patient was operated on in the surgical clinic. The meninges were found to be thickened and light yellow. No tumor was found, but the spinal cord in this region appeared to be thickened like a quill and possibly somewhat discolored. Three weeks later, the patient returned to the clinic. The paralysis of the lower extremities was marked; the Babinski sign was present; the knee reflexes were lost, but the abdominal reflexes were present in the upper quadrant. Sensory disturbances remained the same as before operation.

Treatment with mercurial ointment was tried until November 8, resulting in improvement in the disturbance of pain sensation and in the power of the lower extremities. Movements of the thighs and knees were still weak, but the plantar flexion in the feet had improved. Roentgen-ray treatment of the spine was begun on November 7, coincident with which motor function of the lower extremities improved considerably. The patient was able to walk well without assistance. She showed only slight paresis of the flexors of the lower extremities. The patient was seen twice during the spring of 1922 and again in April, 1924; she was able to take fairly long walks and had been doing her own house work. At this time (April, 1924), the motor functions of the lower extremities were normal; but she had developed pains in the chest, especially on the right side, with loss of pain sensation in a narrow segment on the right side, one or two fingerbreadths below the mammillary plane.

In this case, it is believed that there was an intramedullary tumor. After the operation in 1921 and the preceding summer's treatment, there was slight improvement, both as regards pain disturbance and motor function. Coincident with roentgen-ray treatment, however, in two months the paresis had almost disappeared, and the motor function was almost restored. We cannot reach any other conclusion but that the first slight improvement was due to the decompression through the laminectomy, and that the subsequent improvement was a result of roentgen-ray treatment. The type of tumor present is not known.

Case 3.—A patient, aged 53, came to the clinic in December, 1921, with a history of having had severe pains in the sacral region and in the right thigh in May, 1919. After several days, she was able to get up, but slight pain persisted in the right lower extremity. Two years later, she had numbness of the right leg and foot. In the summer of 1921, there was sensory disturbance in both feet. In September, 1921, there was difficulty in walking, and two months later she developed a perforating ulcer on the right heel.

Examination revealed a slight weakness of the right foot, but mobility elsewhere was normal. There was a Babinski sign of the left foot with loss of the Achilles reflex. The appreciation of movements of the right great toe was not certain. It is evident that sensation for pain was impaired from the fifth lumbar to the second sacral, and for touch from the fifth lumbar to the first sacral segments.

Roentgenograms showed a kyphosis of the upper dorsal vertebrae and scoliosis of the upper lumbar vertebrae, the spinal column elsewhere being normal. Lumbar puncture revealed a pressure of 120 mm., two cells per cubic millimeter, negative Nonne and Wassermann tests. The blood Wassermann test also was negative.

The patient was treated with the roentgen rays, mercuric ointment and potassium iodid, and a month later, appreciation of passive movement in the toe was normal. The Achilles reflexes were present, and the Babinski sign was negative. There was no loss of sensation for touch, but pain was disturbed in the fifth lumbar and first sacral segments. In March, the ulcer in the foot had healed, the area of sensation of pain disturbance had also improved, and the patient left the clinic on March 16.

She was again examined on June 1, at which time she complained of pains in the right leg. The Achilles reflexes were absent, and there was a positive Babinski sign on the left side. The appreciation of movement in the toes was disturbed. There was weakness of plantar flexion in the right foot, and sensation for touch was impaired from the fifth lumbar to the second sacral segment. She was given roentgen-ray treatment for several days, followed by mercury and potassium iodid. In September, there was no weakness of the right foot and no change in the reflexes; there was no definite loss of sensation, but in a part of the fifth lumbar segment, sensation was not certain. She was given additional roentgen-ray treatment, and since that time has not been examined. In January, 1924, she wrote that there had been a relapse of the perforating ulcer. She wrote that on the outer side of the right leg there was "almost no sensibility." She had some pain in the right leg and difficulty in walking.

The diagnosis in this case is an affection of the right posterior horn involving the fibers of the posterior roots in the fifth lumbar and second sacral segments. It is a question whether the symptoms were due to a syringomyelia or to a circumscribed glioma.

The course of the disease showed that roentgen-ray treatment brought about an improvement. It is evident that the progression of symptoms showed the true nature of the disease. The most important result, from a practical point of view, seems to us to be that the perforating ulcer of the foot healed and that the foot had been well for two years.

THE ROENTGEN RAY AND SYRINGOMYELIA

Many authors have reported improvement of syringomyelia through roentgen-ray treatment; Raymond, in 1905, was the first. The names of the later authors who have reported the same (for the most part there were only one or two cases observed, although some of the authors have seen more) are: Mendlici,[1] Gamegna,[2] Ranzoni,[3] Beaugard and

1. Mendlici: J. des Practiciens, 1905, No. 51.
2. Gamegna: Fortschr. a. d. Geb. d. Röntgen **12**:57.
3. Ranzoni: Gaz. med. ital., 1906, No. 46; Ztschr. f. d. Elektr. u. Röntgenkunde **9**:404; Rev. neurol., 1907, p. 224.

L'Hermitte,[4] Beclére,[5] Labeau,[6] Delherm,[7] Desplats,[8] de Noble,[9] Mme. Fabre,[10] Marinesco,[11] Marques,[12] Rimbaud,[13] Lotsy,[14] Roger,[15] Bourguignon and Thomas,[16] Allaire and Denis,[17] Rupin,[18] and Strauss.[19]

French authors have paid especial attention to this question, and these observations would lead one to accept the idea that the symptoms of syringomyelia may be improved by the roentgen rays. Certainly we must *a priori* wait for this to be brought about through a reduction of the newly-formed gliomatous tissue. This opinion is also confirmed by anatomic investigations by Coyon, L'Hermitte and Beaugard in one case in which the patient was treated with the roentgen rays some years ago.

If Case 3 is to be accepted as syringomyelia, one must assume that the anatomic lesions had not developed much farther than to the formation of new gliomatous tissue. Consequently, we can understand that the processes have been reversible—but only for a certain time.

If there are many observations of improvement of syringomyelia by roentgen-ray treatment, this is not true of spinal tumors. Gamegna,[20] in 1906, reported that he had acquired more or less pronounced remission of the symptoms in two cases of spinal tumor (sarcoma?).

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4. Beaugard and L'Hermitte: Semaine méd., 1907, p. 193; Arch. d'Electricite Med., No. 282; Fortschr. a. d. Geb. d. Röntgen. **15**:313 and **17**:252.

5. Beclére: Arch. d'Electricite méd., 1908, p. 473; Fortschr. a. d. Geb. d. Röntgen. **12**:439.

6. Labeau: Arch. d'Electricite méd., 1908, No. 242; Fortschr. a. d. Geb. d. Röntgen. **13**:63.

7. Delherm: J. d. sc. méd. de Lille, 1909.

8. Desplats: J. de radiol. **3**, p. 26; Ztschr. f. Elektr. u. Röntgenkunde **11**: 409, 1909.

9. De Noble: Progrés méd., Dec. 18, 1909.

10. Fabre: Progrès méd., 1910, p. 216.

11. Marinesco: Arch. d'Electricite méd., 1910, No. 299; Fortschr. a. d. Geb. d. Röntgen. **16**:404.

12. Marques: Progrès méd. **23**:6; Fortschr. a. d. Geb. d. Röntgen. **17**:253.

13. Rimbaud: Arch. d'Electricite méd. **11**:335, 1912.

14. Lotsy: Arch. d'Electricite méd. **11**:238, 1912.

15. Roger: Rev. neurol. **1**:175, 1913.

16. Bourguignon and Thomas: Rev. neurol., 1913, p. 471.

17. Allaire and Denis: Contribution a l'etude d' traitment radiotherapeutique de la syringomyelia, These Montpellier, 1913; ibid. **1**:103, 1914.

18. Rupin: Strahlenterapie **2**: 402.

19. Strauss: Bull. et mém. Soc. méd. d. hôp. de Paris **38**:387, 1922; Zentralbl. f. d. Neurol. u. Psychiat. **29**:281.

20. Gamegna: Arch. f. Psychiat. **57**:920, 1917; Deutsch. med. Wchnschr., 1916, p. 222; München. med. Wchnschr., 1918, p. 521; Ztschr. f. d. ges. Neurol. u. Psychiat. **76**:81, 1922.

Saenger,[21] in Hamburg, reported a case of neuro-epithelioma gliomatodes in which the patient was first operated on but the tumor not totally extirpated; later, there was improvement with the roentgen-ray treatment. Saenger saw two other cases of tumor in which the patient would not permit operation and in which the objective symptoms were not improved by roentgen-ray treatment.

Gerhardt has tried roentgen-ray treatment after extirpation of an endothelioma durae matris, but the results were not published. Recently, Fischer has concluded that an intramedullary tumor improved with roentgen-ray treatment. He tried similar treatment in other cases, without improvement.

Our observations show that roentgen-ray treatment has produced improvement in the symptoms of a diffuse myxosarcomatosis of the meninges; also in a case of intramedullary tumor the nature of which was unknown, and perhaps in a case of glioma.

While we are unable to solve the question, we call attention to this treatment, which seems to be worthy of a trial when operation for one reason or another is not advisable.

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21. Saenger: Arch. Psychiat. **57**:920, 1917; Deutsch. med. Wchnschr., 1916, p. 222; München. med. Wchnschr., 1918, p. 521; Ztschr. f. d. ges. Neurol. u. Psychiat. **76**:81, 1922.

PSYCHIC REPRESENTATIONS OF MOVEMENT AND POSTURE

THEIR RELATION TO SYMPTOMATOLOGY *

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In a series of previous studies [1] I have formulated a conception of the efferent nervous system, founded on the existence, as I believe, of dual pathways subserving respectively the functions of motion and posture. According to this theory, the kinetic mechanism consists of a series of systems which are concerned with the production of movement, while the static mechanism represents a coextensive series of neural systems which are concerned with the maintenance of posture. All of the complicated phenomena of motility are dependent on these twin mechanisms, which function together in harmony.

In accord with this duality of function, striated muscle fiber is composed of two distinct substances, one subserving a contractile, the other a postural function. The sarcostyle is the contractile portion of the muscle fiber and represents the myokinetic mechanism; the sarcoplasm is the postural constituent and represents the myostatic mechanism.

In disorders of motility, as in normal motility, both systems participate, although it is possible to indicate one or the other as essentially involved. Involuntary movements, like chorea, myoclonus, tremor and convulsive seizures are of kinetic origin. Myotonia and catalepsy are disorders of the posturing mechanism, as are also the characteristic symptoms of cerebellar disease.

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* Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June, 1924.

1. Hunt, J. Ramsay: The Static and Kinetic Systems of Motility, Arch. Neurol. & Psychiat. **4**:353 (Oct.) 1920; The Static or Posture System and Its Relation to Postural Hypertonic States of the Skeletal Muscles, Spasticity, Rigidity and Tonic Spasm, Neurol. Bull. **3**:207 (June) 1921; The Dual Nature of the Efferent Nervous System. A Further Study of the Static and Kinetic Systems, Their Function and Symptomatology, Arch. Neurol. & Psychiat. **10**: 37 (July) 1923; Dyssynergia Cerebellaris Myoclonica (Primary Atrophy of the Dentate System). A Contribution to the Pathology and Symptomatology of the Cerebellum, Brain **44**:490, 1921; The Relation of the Cerebellum to the Static System and Its Rôle in Posture-Synergy, J. Nerv. & Ment. Dis. **60**, No. 4, October, 1924; The Statesthetic and Kinesthetic Components of the Afferent Systems, Abstr., Arch. Neurol. & Psychiat. **8**:311 (Sept.) 1922; Static Seizures in Epilepsy. A Type of Epileptic Paroxysm Characterized by Sudden Loss of Postural Control, Revue neurol. **11**, No. 3, September, 1924.

This principle of duality of motor function is also applicable to the vegetative nervous system. Here the parasympathetic subserves a kinetic and the sympathetic outflow a static function; for the unstriped contractile fiber, like the striped variety, is composed of two distinct substances, fibrillae and sarcoplasm, which subserve respectively primitive types of motion and primitive types of posture.

The evidence bearing on this question is considered in my previous communications, and it is interesting to note that the operative procedure recently advocated by Hunter [2] and Royle [3] is confirmatory of this point of view.

The present study is concerned with the static and kinetic representations of these two mechanisms of the efferent system in the psychic sphere, for even in this complex realm there are evidences of a dual function.

KINETIC AND STATIC REPRESENTATIONS OF THE EFFERENT SYSTEM IN THE PSYCHOMOTOR SPHERE

In the higher motor activities of the cerebral cortex the simpler elements of motility undergo various combinations which are expressed as purposive acts and postures. In this realm are included such disorders of motility as aphasia, apraxia, perseveration and stereotyped movements and attitudes.

This is the field of acquired motility, and one which may be indefinitely expanded with experience and the increasing demands of civilization. Purposive acts are the natural expression of motility at the psychic level and present every conceivable variation, according to the habits and education of the individual. They represent ideas which are transformed and expressed in action.

In symptomatology a loss of this function is called apraxia, a term signifying an incapacity to execute purposive movements notwithstanding the preservation of muscular power, sensibility and coordination. In this disorder the defect is situated in those areas of the cerebral cortex in which ideas are formed and expressed as purposive movements and postures.

Among the psychomotor activities of this description may be mentioned the simple acts of everyday life; for example, lighting a cigaret, closing a door, the use of a knife and fork, indeed all of those arts and crafts which are acquired by training and experience, the imprint of which remains fixed in memory and may be recalled and transformed

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2. Hunter, J. I.: Postural Influence of the Sympathetic Innervation of Voluntary Muscle, M. J. Australia **1**:861, 1924; Brain **47**:261, 1924.

3. Royle, N. D.: A New Operative Procedure in the Treatment of Spastic Paralysis, M. J. Australia **1**:77 and 125, 1924; Brain **47**:275, 1924.

into their corresponding actions and attitudes. In this sense the idea of an action or of an attitude is dependent for its expression on a psychokinetic or psychostatic representation in the same manner that a reflex movement or a reflex posture is dependent on its corresponding archeokinetic or static mechanism.

Two great clinical types of apraxia may be recognized. One is due to a loss of the memories of objects—an amnesic apraxia. In the other, although memory is undisturbed there is an inability to perform the act, and this constitutes the motor apraxia. In this sense, certain motor disorders of speech and writing may be mentioned as apraxias of a higher order. Motor apraxia is therefore a paralysis of purposive movements and is the analogue in the psychomotor sphere of the other cardinal types of motor paralysis which are characteristic of lower levels.

In apraxia, a peculiar manifestation has been described by some writers as preservation, which appears to have a direct bearing on the question of psychostatic and psychokinetic representations in the cerebral cortex.

Kinetic Perseveration.—Perseveration in motor apraxia may be defined as the continued repetition of a given movement complex when another act is intended. The repetition does not occur spontaneously but only when some new act is intended and in place of that act. This symptom of cortical disease has been termed intentional perseveration by Liepmann. Another form of perseveration is that in which the patient continues making a particular movement or movement complex, showing an inability to inhibit the movement once it has been initiated. Both of these types are well recognized in the symptomatology of nervous and mental disease.

It is interesting to note that perseveration may also occur in the sphere of speech and writing.

Static Perseveration.—In addition to the types which are characterized by a disorder of movement, there is another form of perseveration which, I believe, is related to the posture sphere, and which is characterized by a cessation or fixation of movement. In this form the patient comes to a standstill during or at the close of the performance of a given act. This may appear as a local or general manifestation, but in either case there is a fixation in a particular attitude which may be indefinitely prolonged. Cases presenting this symptom have been described by Pick,[4] Kroll,[5] and Coriat.[6]

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4. Pick: Studien über motorische ataxie, Liepzig & Wien, 1905

5. Kroll: Beiträge zum studien der Apraxie, Ztschr. f. d. ges. Neurol. u. Psychiat. **11**:315, 1910.

6. Coriat: The Psychopathology of Apraxia, Am. J. Psychiat. **22**:65, 1911.

According to Wilson and Walshe,[7] the repetition of a given movement in place of another, and the continued repetition of a given movement should be known as active perseveration, whereas the cessation of action which results in the maintenance of an attitude, either in the middle or at the end of a given movement complex should be known as passive perseveration. These terms they suggest in preference to tonic and clonic, which were used by Liepmann[8] in his description of the same types.

The terms kinetic and static perseveration, I believe are even more descriptive as referring to the specific type of motor representation involved in the disorder.

In Pick's study of motor apraxia, cases are described which have a special bearing on this problem. One of his patients, asked to take a drink out of a jug on the table, put his lips and face into the mouth of the jug and remained so practically without moving. Another patient, asked to light a candle, held the match alight in her right hand, near the wick, quite immobile, until the match burned her fingers. These are examples of static perseveration. There is a premature postural fixation of a purposive movement before the purpose of the movement has been carried out.

In kinetic perseveration, the patient continues uninterruptedly making a particular movement or movement complex. Liepmann's patient, for instance, having begun to write certain words was unable to stop, and continued scribbling in senseless repetition. Campbell's[9] patient "continued to shake hands for an unusual length of time." A patient observed by Wilson, writing her name "Winnie" wrote Winninninninn . . . indefinitely. A patient of Breukink's,[10] peeling potatoes, continued peeling apparently unable to cease.

Both of these forms therefore represent a cortical disorder of motility and are dependent on the release of static and kinetic representations at this level of the nervous system. One is a kinetic perseveration and the other a static perseveration, and both are referable to an organic disorder of the efferent system in the psychomotor sphere.

PSYCHOKINETIC AND PSYCHOSTATIC MANIFESTATIONS OF HYSTERIA

Psychostatic and psychokinetic types of reaction may also be recognized in the various paroxysmal crises of major hysteria.

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7. Wilson and Walshe: The Phenomena of Tonic Innervation and Its Relation to Motor Apraxia, Brain **37**:199, 1914.

8. Liepmann: Ueber Störunger des Handelns bei Gehirn Kranken, Berlin, 1905.

9. Campbell, MacFie: Agraphia in a Case of Frontal Tumor, Rev. Neurol. & Psychiat. **9**:289, 1911.

10. Breukink: Ueber Patienten mit Perseveration und Asymbolischen und Aphasischen Erscheinungen, J. Psychiat. u. Neurol. **9**:113, 1917.

The classical types of hysterical disorders of motility—convulsions, chorea, myoclonus and tremor—are all examples of the release of kinetic representations. These motor phenomena are all of psychogenic origin and are initiated by unconscious mental mechanisms or by dissociations of cortical function. They often closely simulate other kinetic manifestations which originate in lower motor mechanisms.

Disorders of the posture mechanism may also occur in major hysteria, an important example of which is catalepsy. This may be partial or general, and is characterized by a peculiar stiffness of the musculature and a waxy resistance on making passive movements. This is the flexibilitas cerea which fixes the limb in posture as soon as movement ceases, and which may persist for a long period of time. A certain resemblance between myotonia and catalepsy may be mentioned and is of special interest, as both of these symptoms, I believe, are referable to the static system.

STEREOTYPED MOVEMENTS AND ATTITUDES IN DEMENTIA PRAECOX

In other disorders of cortical function, the occurrence of kinetic and static types of reaction are also evident in the symptomatology.

In dementia praecox, for example, with its widespread disorder of psychic functions, many peculiar disturbances of motility are described. When a disturbance of the psychostatic mechanism occurs, there is a fixation of the muscles in certain postures or attitudes, which may persist for weeks, months or even years. In the kinetic sphere various stereotyped movements occur which are so common in the catatonic form of this disorder. These movements may be repeated countless times over long periods of time.

According to Kraepelin, stereotypy is a persistent fixation of certain muscle groups or a repetition of the same movement. In the first case, the patient in spite of all external influence maintains the same attitude for weeks, months or years, with scarcely any change. He may stand in the same posture, often a very uncomfortable one, or kneel on a certain spot. The facial expression is stiff and masklike, and the winking reflex diminished or obliterated.

Stereotyped movements are naturally much more varied in character. Among these may be mentioned, the somersault, rhythmical clapping, making the sign of the cross, jumping, falling, rolling and creeping on the ground, bizarre arm movements, pulling the clothing and hair, bowing, balancing, rocking movements and grinding the teeth. These movements may be repeated numberless times for weeks and months.

It is therefore apparent that in organic disease of the cerebral cortex, in hysteria and in dementia praecox, disorders of the psychokinetic and psychostatic mechanisms may occur similar in nature to those at lower levels.

These disturbances of movement and of posture indicate that while the function of the efferent system becomes more complex with the progress of evolution, the same representations of function which were encountered in the lower are also present in the highest psychomotor levels of the central nervous mechanism.

I shall now consider purely psychic representations of the efferent system, for in this intricate and complex realm there are evidences of a duality of function—the analogues of movement and posture.

KINETIC AND STATIC REPRESENTATIONS OF EFFERENT NERVOUS SYSTEM IN PSYCHIC SPHERE (THOUGHT AND BELIEF)

The various functional divisions of the efferent nervous system are largely schematic, and no exact line of demarcation separates them. Each physiologic level merges into the higher level by imperceptible gradations, and all of the great physiologic motor systems are but parts of a highly integrated whole, which represent in the efferent sphere the various stages of evolutionary development. Reflex movements, automatic-associated movements, isolated synergic movements and purposive movements with their corresponding postures represent successive steps in the development of the great expressive functions of the nervous system.

I shall try to show that this conception of the movement and postural components of the efferent nervous system is also applicable in the purely psychic sphere. If the conception of a static and kinetic system is true of the motor and psychomotor levels, it should also hold for the purely psychic components of the expressive mechanism.

Psychic processes contain both sensory and motor elements in complex and highly integrated combinations; and in the structure of mind there is represented not only the receptive systems but also their corresponding expressive mechanisms. For these two great functions have always existed side by side in the slow and tedious processes of evolution, and evidences of a sensorimotor mechanism are present in mental function as in the lower levels of the central nervous system.

Perception in the psychic sphere does not differ in its nature from the sensation of lower centers, but only in the complexity of the sensory stimuli involved. Both are sensory, both are receptive, and both serve the purpose of bringing the organism into relation with the outer world. This is true of all modalities of sensory stimuli.

I would maintain the same thesis for the efferent or expressive mechanism in the psychic sphere.

The Psychic Representation of the Kinetic System (Thinking).—The highest level of the nervous system is the psychologic level in which percepts and concepts, represented by language symbols, play the predominant rôle. This sphere includes such purely intellectual processes as perception, conception, judgment, imagination and reason.

In this great realm of psychic activity, nerve cells and nerve fibers represent the structural basis of function, as in lower levels, differing only in their quality and the complexity of their structure and association. In the psychic sphere, as in lower levels, we encounter the representations of motion and sensation, excitation and inhibition, coordination and association, only in more complex and more highly integrated combinations.

Our present knowledge of the correlation of structure and function in the central nervous system would indicate that these purely psychic functions are subserved by the supragranular layers of the cerebral cortex and their various association systems. Destroy those regions in whole, or in part, and there is a corresponding loss of mental function.

This region subserves the function of thought, which I believe should be regarded as the highest expression of the efferent mechanism. I would designate the systems of this realm which are concerned with the function of thought as the noetic systems.[11] This term indicates their special relation to the act of thinking and distinguishes them from the kinetic systems proper of the efferent mechanism. For while thought is essentially a representation or sublimation of movement, its corresponding kinetic expressions are inhibited. In accordance with this view, the act of thinking is the analogue and representation of movement at lower levels.

In common with the higher forms of movement, thought is voluntary and purposive and is the highest expression of adaptation of the individual to the outer world.

The development of motility therefore represents the adaptation of the human organism to the world of reality in terms of movement and posture. The development of thought represents a still higher relation of the human organism to the world of reality which is represented in language symbols, and at the psychologic level the sensory and the motor patterns are the language mechanisms. Therefore the act of thinking represents an activity in the sphere of language symbols and their combinations which has been acquired by education and experience.

If thought is purposive and an adaptation of the human organism in the mental sphere, what then is its object? The object of thought is the attainment of belief in response to the stimulus of doubt. A doubt arises in the mind which stimulates inquiry, which is thought, the object of which is to reach a mental state that may be expressed by such terms as decision, conclusion or belief. Thinking, therefore, is an effort to reach the mental state that these terms indicate, and which gives rise to a feeling of rest or relaxation after the activity of thought.

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11. Noetic—from the Greek "nous" signifying thought.

A decision is always an essential step in the realm of any purposive action, which may or may not be given motor expression, depending on the will of the individual. The inhibition of action after decision permits deliberation and the formation of beliefs which are the basis of future action. The control and inhibition of action in thought shows the great gulf separating the intellectual activities from the other purely somatic expressions of man.

One of the essential factors in the operation of thought is inhibition, which suppresses all corresponding expressive manifestations. Each functional level of the nervous system exercises a power of control over lower levels. The psychokinetic, neokinetic and paleokinetic systems all inhibit subordinate mechanisms. In this respect the psychic level resembles the other great physiologic levels of the efferent mechanism. The purely thinking or neotic systems possess to a high degree this power of control over lower levels by virtue of the general law of inhibition which regulates all of the levels of the efferent system.

The various forms of reasoning, then—reasoning from particulars, inductive and deductive reasoning—are but modes of intellectual activity which are essentially motor in their representation but with inhibition of the corresponding modes of expression.

The Psychic Representations of the Static System (Belief).—I have already stated that the object of thought is a decision or a belief. Following the activity of thought, which is inquiry, a conclusion is reached which is followed by mental relaxation or rest. This represents the fixation of thought in terms of a decision, a conclusion, or a belief, and bears a certain analogy to the fixation of posture after movement. The common expression, an attitude of mind, therefore, has a deeper significance than is usually given it, as it correctly describes the starting point of thinking and its termination.

Our minds are composed of innumerable beliefs, large and small, systematized and unsystematized, which have resulted from thinking. They represent the mental attitude assumed after thought, and form starting points for further thinking. In this respect they again resemble postures, as movement always starts from and terminates in posture.

Therefore, as thought is kinetic and the analogue of movement, belief is static and the analogue of posture in the psychic realm.

It is here interesting to quote from Charles H. Peirce,[12] the distinguished philosopher of pragmatism, who in interpreting certain relations of mind, has presented, from another point of view, the hypothesis which I have just expressed.

> But the soul and meaning of thought, abstracted from the other elements which accompany it, though it may be voluntarily thwarted, can never be made

12. Peirce, Charles H.: Chance, Love and Logic, 1923, p. 40.

to direct itself toward anything but the production of belief. Thought in action has for its only possible motive the attainment of thought at rest; and whatever does not refer to belief is no part of the thought itself.

And what, then, is belief? It is the demi-cadence which closes a musical phrase in the symphony of our intellectual life. We have seen it has just three properties; first, it is something that we are aware of; second, it appeases the irritation of doubt; and third, it involves the establishment in our nature of a rule of action, or—say for short, a habit. As it appeases the irritation of doubt, which is the motive for thinking, thought relaxes, and comes to rest for a moment when belief is reached. But, since belief is a rule for action, the application of which involves doubt and further thought, at the same time that it is a stopping-place, it is also a new starting-place for thought. That is why I have permitted myself to call it thought at rest, although thought is essentially an action. The final upshot of thinking is the exercise of volition, and of this thought no longer forms a part; but belief is only a stadium of mental action, an effect upon our nature due to thought, which will influence future thinking.

This conception of the relation of the processes of thought to movement which I have expressed is not wholly new in psychology. To some extent this point of view was foreshadowed in the ideomotor theory, so well stated by William James,[13] but above all in the more recent formulations of the behavioristic school.

Watson[14] has recently summarized the behaviorist's conception of thinking as follows:

What Man Is Doing When Not Overtly Acting.—With a highly specialized organism like man, even careful observation often fails to show any overt response. A man may sit motionless at his desk with pen in hand and paper before him. In popular parlance we may say he is idle or "thinking" but our assumption is that his muscles are really active and possibly more active than if he were playing tennis. But what muscles? Those muscles which have been trained to act when he is in such a situation, his laryngeal, tongue, and speech muscles generally. Those muscles are as active and are carrying out as orderly a system of movements as if he were executing a sonata on the piano; they are doing it well or ill, depending upon the training he has had along the particular lines which engage him. While we cannot at present watch the play of this implicit stream of words, there is no reason for hypothecating a mystery about it. Could we bring "thinking" out for observation as readily as we can tennis playing or rowing, the need of "explaining" it would disappear. We shall see later that efforts have been made to bring such responses under experimental control. But entirely apart from our present unreadiness to make observation on implicit habits, we find a certain way of arriving indirectly at the same end; implicit language habits, by methods which we shall study, come to issue finally in overt action. By watching the easily observable explicit habits and instincts of an individual keenly enough, and for a sufficient stretch of time, and under varying enough conditions, we can obtain the necessary data for most psychological requirements.

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13. James, William: Psychology **2**:522.

14. Watson, J. B.: Psychology from the Standpoint of a Behaviourist, J. B. Lippincott Co., 1924, p. 15.

Here then is a definite statement of the relation of thought to motion. Thought, according to this view, is subvocal speech. All thought is merely an implicit language form of action which may be demonstrated by exact experimental method applied to the motor mechanisms, and during thought there is a corresponding flow of motion into the vocal or other expressive mechanism of the body.

While admitting that in many cases thought is expressed by simultaneous speech (as in thinking aloud), and by expressions of the face, attitudes and gestures, this by no means always follows. In my theory of thinking, the intricate and elaborate language representations of movements exist, and yet all motor expression of them may be inhibited. Indeed, this is the very essence of pure thought and what unquestionably occurs in the higher and more disciplined types of mind. It is this part of the behaviorist's conception of thought which has aroused the greatest antagonism among psychologists.

If, however, we conceive of pure thought as an implied language activity all expression of which is inhibited, according to the laws of inhibition elsewhere in the efferent system, this obstacle is removed. The mechanism of inhibition is so important a factor in all neural processes that it is not surprising to encounter it here in the highest levels of the central nervous system. And in conformity with the general law of the nervous system that each higher level has the power of inhibition over lower levels, its appearance in the psychic realm is a natural outgrowth of evolution in the interests of deliberation and control.

In a previous study,[15] I have considered the question of the neural mechanism underlying inhibition in the central nervous system, and I have presented certain evidence showing that this function is subserved by small ganglion cells of the Golgi type (Type 11). It is interesting to observe that these small cells are particularly abundant in the cellular layers of the cerebral cortex which subserve the functions of thought (the noetic system).

The Psychic Representations of the Efferent Nervous System and Their Relation to Symptomatology.—In my earlier studies of the dual nature of the efferent system, it was shown that certain disorders of movement were referable to the kinetic system, while disorders of the posture mechanism were related to the static sphere. Evidences of such dissociation in the morbid physiology of movement were shown to exist at all of the great physiologic levels of the central nervous system. A similar dissociation was noted in the various

15. Hunt, J. Ramsay: A Theory of the Mechanism Underlying Inhibition in the Central Nervous System, Arch. Neurol. & Psychiat. **11**:418-431 (April) 1924.

psychomotor manifestations of the cortical realm. I believe that there is evidence of a similar dissociation in the purely psychic sphere.

Disorders of Thinking (Kinetic Component of Thought).—As thinking is essentially expressive and a manifestation of the efferent nervous system, one would expect in the dissociations of function resulting from mental disease a disturbance of the processes of thought similar to the disorders of movement at lower levels.

In the lower levels of the kinetic system, two types of motor disorders may be recognized: one is a loss of motor function which is indicated clinically by paralysis; the other is a discharge of motor activity which reveals itself as convulsions, spasms, spastic states and the other clinical expressions of hyperkinesis.

Loss of motor function may express itself as spinal, extrapyramidal or pyramidal types of palsy, with corresponding loss of reflex, automatic-associated or isolated synergic types of movement. We have already considered the paralysis of purposive movement in the psychomotor sphere under the term apraxia.

The counterpart of this symptom in the mental sphere is paralysis of thought with consequent loss of judgment and the reasoning faculties. This is present in those widespread disorders of the cerebral cortex which result in dementia.

An excessive display of motor function at lower levels is manifested by the various convulsive disorders of motility at the spinal, striatal and cortical levels. Various convulsive states related to the psychokinetic system have already been presented.

At the psychic level an analogous disorder of thinking also occurs, which is manifested by an exaggerated activity of thought. The flight of ideas of the manic state is of this nature and represents an increased activity and discharge of thought which bears a certain analogy to the general convulsive manifestations of lower levels. There is an excessive and involuntary discharge of neural energy in terms of thought. This in the realm of thought is the analogue of hyperkinesis in the motor sphere. In the manic state there is also a considerable release of psychomotor activity as well, which is manifested in excessive activity of speech and action.

The so-called "crowding of thought," a symptom occasionally observed in schizophrenia, is also a manifestation of excessive discharge in the thinking mechanism. Bleuler[16] describes this peculiar disorder of thought:

> In such cases the content subjectively seems rich and varied, and gives the impression of a continuous mental stream. But if the attempt is made to

16. Bleuler: Textbook of Psychiatry, New York, The Macmillan Company, 1924, p. 80.

penetrate a little more deeply, one regularly gets the impression that the patients are forced to think always the same thoughts. The crowding of thoughts is in most cases connected with a disagreeable feeling of exertion.

A more limited manifestation of the higher activity of thought release is to be found in certain of the fixed ideas and obsessions of the compulsion neurosis. These are characterized by an involuntary stereotyped activity of thought. The so-called questioning and reasoning manias are of this type. Many of the fixed ideas force themselves into action, but others remain permanently subjective, and all corresponding motor expressions are inhibited. It is a disorder of the thought mechanism alone.

Such purely psychic manifestations of the compulsion neurosis are the analogue of psychogenic tics, in which the impulsive thought at once passes into its corresponding motor expression. In the compulsive thought of the purely reasoning type, however, the disorder is of the thinking mechanism alone.

Disorders of Belief (Static Component of Thought).—A disorder of the static mechanism of lower levels of the central nervous system is characterized by the inability to regulate the postural function of the body.

There may be an inability to relax a postural fixation, as in myotonia; or the ability to fix or synergize movements in terms of posture may be lost, as in cerebellar disease. Mention has already been made of the disorders of this function in the psychomotor sphere, under the heading of static perseveration, stereotyped attitudes and catalepsy.

In the purely psychic sphere, a morbid inability to fix the processes of thought in terms of a decision is sometimes observed in certain forms of mental disease. This manifestation is not uncommon in compulsion neurosis. The doubting mania is of this type, in which the individual is obsessed by doubt and indecision. This would appear to be a disorder in the static sphere of mind, characterized by a loss of the ability to fix the processes of thought in decision.

In severe forms of mental disease, the fixed delusion may in certain cases also represent a disorder of the static representation of thought, a disorder in the sphere of belief. Here a belief is formed and remains fixed and stereotyped, influencing in a deleterious manner the future thinking activities of the individual. The reasoning from a false belief is the very essence of the paranoid reaction. The thought processes may be logically quite correct, but the starting point or belief is wrong. It is, in a sense, the analogue in the sphere of belief of the stereotyped attitude in the psychomotor sphere.

Therefore, in the psychic sphere certain disorders, both of thinking and belief, may be recognized in the symptomatology of mental disease, which bear a certain analogy to the disorders of movement and of posture at lower levels.

CONCLUDING REMARKS

The present study concludes the evidence on which I base my conception of the dual nature of the efferent nervous system.

In these investigations I have laid the foundation for the hypothesis that kinetic and static components of motility represent a parallelism of structure and function at the various levels of the efferent mechanism; and that the motor systems of the organism, like those of sensibility, may be resolved into more than one component subserving different types of function.

The conception of a static and kinetic mechanism functioning together in the interest of motion and posture is confirmed in many fields of research—in histology, physiology, biology, chemistry, comparative anatomy and neurology. From these sources the evidence is striking and to my mind conclusive. In this paper I have indicated the value of this conception even in the realm of psychology.

The development of these two great types of motor function represents the adaptation of the organism to surrounding physical forces. The kinetic system is concerned with movement of the organism, while the static system maintains the organism in postural fixation. Both functions are necessary in the adaptation of the organism to surrounding physical forces and the purposive functions of life.

In the central nervous system of man, four great physiologic divisions of the efferent mechanism may be recognized, which correspond to the various evolutionary epochs in the development of motility. These are represented by reflex, the automatic-associated, isolated-synergic and purposive types of movement and posture. In the psychic realm, thinking and belief are the analogues of movement and posture.

In this conception of the efferent mechanism, I would again emphasize the harmonious interaction and functional unity of all these systems in the total reactions of the individual.

The simplest types of movement and the highest expressions of thought I would regard as two extremes of the great expressive functions of the human organism. These two extremes are united by a series of ascending functional levels which are a recapitulation of evolutionary history and the motor functions of life.

In the presentation of this conception, I quite realize that many of my conclusions must be regarded as still in the realm of speculation. But this is true of all hypotheses, even of those resting on a secure foundation. It represents, however, a new method of approach to the investigation of the important problem of the efferent mechanism of life.

SEQUELAE OF EPIDEMIC ENCEPHALITIS WITHOUT ANY PRECEDING ACUTE ILLNESS (CHRONIC ENCEPHALITIS)

REPORT OF CASES *

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The determination of the cause of acute brain disease, especially when the patient is first seen weeks or months after the onset, has become more difficult since the reappearance of epidemic encephalitis in 1917, because, seemingly, new and certainly unfamiliar, clinical pictures are frequently presented to us. During the epidemic, once the disease became known, the diagnosis was, or seemed to be, easy; given a patient ill for a few days with fever, transitory diplopia, much or little headache and somnolence, and the diagnosis was made. Soon it was noted that in many patients instead of somnolence there was delirium, or the clock was turned round, and the patient slept all day and was delirious all night. After a time, it was learned that there were characteristic sequelae appearing at varying times after the acute attack. Finally, and I think it is the last complicating factor in the study of this disease, cases began to appear, and with increasing frequency, in which the sequelae appeared without any preceding acute illness. The cases I report illustrate this point and also some of the many difficulties in differential diagnosis.

REPORT OF CASES

CASE 1.—*History.*—This case shows the influence of soil, the kind of protoplasm one is born with, in determining the results of acquired disease. If the youth had had a different heredity, he would have presented a different clinical picture, because different protoplasms react differently to the same stimuli. Had the physicians who examined him before he came under my care learned his family history before making their diagnoses, there would not have been such great differences in their opinions. Forming their opinions on a mere cross section of his life, his condition on one day, one diagnosed hysteria, another dementia praecox, a third high grade imbecility and the fourth petit mal.

He came of highly intelligent but uneducated German-Russian Jewish stock. His mother was very neurotic, indeed hysterical, and his father was subject to outbursts of causeless anger, followed by periods of depression. Neither parent knew how to train and guide a boy born, as this boy was with abnormal tendencies.

The patient during childhood and early boyhood was bright but neurotic, and presented no marked abnormalities in conduct or behavior until he was 17 years old, when he began to have a respiratory tic, for which I could find

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* Read before the Philadelphia Neurological Society, Oct. 24, 1924.

no exciting cause. At first he had many attacks daily, of rapid, noisy expiration lasting a minute or two. Some months later, his nose was fractured, and the resulting obstruction increased the severity but not the frequency of the tic. The nose was operated on, and soon after he passed through an attack of scarlet fever. Several months later, the respiratory tic ceased. The cause of the cessation I do not know, but I am sure it was not the curative effect of the fever. He returned to high school when 18 years old (January, 1923), but did not do well and soon began to have "drowsy spells." The teacher, two months after his return, notified his parents that he would either have to keep awake or leave the school. No one suspected illness; every one, like the teacher, assumed laziness. During the Easter vacation, almost three months after somnolence began, he suddenly became acutely ill with sore throat, vomiting and fever, and two days after the onset delirium appeared. At the end of a week, he seemed to be well, and returned to school, which he attended until the end of the school year, but he did not do well. The periods of sleepiness continued, and he failed in his examinations.

He was brought to me when 19 years old because of personality changes. His parents stated that from the time the tic began they had noticed alterations in his conduct, which they thought indicated viciousness. Previous to its appearance he had been a bright, happy, ambitious and industrious school boy. He grew lazy, surly, had outbursts of causeless anger and became disrespectful toward his parents. They verified the history of attacks of sleepiness, and rapid breathing and were positive that the acute febrile attack, mentioned above, occurred months after the other symptoms.

Examination.—The most striking thing in his appearance was the facial rigidity. He had the facies of paralysis agitans. The skin was oily over the entire body, and he had facial acne. During my first examination, he had an attack of hysterical rapid breathing. Neither attitude, excluding the face, nor gait suggested paralysis agitans, but at times he showed local catalepsy. If I put either arm in any position, he would hold it so for an indefinite period, and then it would slowly drop to the side. He was pleasant in manner toward me (this changed later) but was somewhat childish, making me suspect the beginning of an adolescent dementia.

Treatment and Course.—I sent him to the Infirmary for Nervous Diseases for observation. While there, his mental symptoms became marked. He had periods of surliness, was childish in manner and took interest in nothing. He had no periods of drowsiness; on the contrary, he slept poorly. He had several attacks as follows: he would hold his breath, become blue in the face, stand with eyes wide open staring into space, extend the arms and fingers stiffly, remain rigid and speechless for about half a minute, then relax and become normal. He was not unconscious during the attacks, and they were manifestly hysterical. I sent him away under the care of a skilled companion, but he rapidly became more demented and unmanageable. He would refuse to get out of bed, several times attacked the nurse in fits of causeless anger, talked impertinently to strangers, and would make suggestive and even obscene remarks to passing women. He continued to have attacks of rapid breathing and to show local catalepsy at times. Throughout the whole period of my care of him, he realized that there was something wrong mentally with him, except at certain short periods when he became dull and stupid, not somnolent, if left alone, but angry, abusive, profane and obscene if ordered to do anything. When I last saw him, six months after my first examination, the

symptoms of paralysis agitans had increased, his facial muscles were more rigid, there was a fine tremor of both hands, and he had begun to stoop at the shoulders.

My final conclusion was that the paralysis agitans syndrome was the result of epidemic encephalitis, but that the dementia would have appeared sooner or later in any event, indeed had already existed before.

Case 2.—Case 2 is interesting only because in October, 1924, three years after his wife began to realize he was slowly becoming sick, the patient presented a good picture of the paralysis agitans type of epidemic encephalitis.

He had influenza during the great epidemic in October, 1918, at which time he was 25 years old. There was rapid convalescence and no immediate sequel. One year later, and while, it is alleged, he was perfectly well mentally and physically, he married. He was a steady worker in a dye house and took good care of his wife. Two years later (1921), he complained of pain in the back of his neck, but he had no acute symptoms, no fever, diplopia or somnolence. Some months later, he began to be very slow in all his movements, and he told his wife that he was not able to do his work because he could not move quickly enough. Looking back (in October, 1924), she dated the fixedness of his facial expression and the tremor, which is confined to the right arm, especially the hand, and right leg, to that time. She could not give an exact date, but stated, positively, that he had no acute illness, and showed no symptoms of disease until more than two years after their marriage. He now has frequent attacks of somnolence. She is positive that it was the last symptom to appear, and it began about six months ago. Mentally, he is normal except that thinking is somewhat slow. This slowness is more apparent than real and depends largely on his muscular slowness. His judgment is good, and he realizes the serious consequences his inability to work will have on himself and his wife. His moral and emotional sides show no signs of degeneration. Syphilis is excluded both by a negative history and by laboratory tests.

That influenza may have the same sequelae as encephalitis is well known. Indeed, there is a close relationship between the two diseases, and this case illustrates it.

Case 3.—This case is interesting because of the different diagnoses. One physician diagnosed the condition as residual left hemiplegia, and, in spite of its resemblance to a postencephalitic paralysis agitans syndrome, he believed the cause to be a specific thrombosis. My own opinion is that it is a pure case of epidemic encephalitis.

The patient was born in Italy, is 35 years old and an acrobat. He had a chancre when 33 years old, and received twenty-five intravenous injections followed by twenty-six injections of mercury. One year after the primary sore, he was speechless for several months, but he could hear and read understandingly and, after a period of good health, during the summer of 1922 (when 34 years old), he slowly developed weakness of the left hand and arm, and still later, similar weakness of the left leg accompanied by a fine tremor, first in the hand and later in the leg.

The blood and spinal fluid, examined several times since 1921, had always been negative.

At the time of my examination (Sept. 19, 1924), he had the facies and attitude of paralysis agitans. There was a constant fine tremor of the left hand (of three years' duration). A seeming head tremor was really transmitted from the shoulder. Tremor was absent in the right arm, leg and tongue. The knee

reflex was normal and the chin reflex was present. He dragged the left leg somewhat when walking. The fingers of the left hand were flexed in the pill-making position. The palpebral fissure was wide (one time he had the hyperthyroid look).

He was sent to Holmesburg on June 24, 1924, but returned in August because he had become quarrelsome and even violent.

Comment.—This case reminds me of the cases of slowly oncoming hemiplegia reported by Dr. Mills before this society years ago. At the time the report was made, I was inclined to believe that they were aberrant cases of paralysis agitans. Looking back, I believe that they were sporadic cases of epidemic encephalitis. It is probable that the organism causing the disease never disappears entirely, but that at irregularly recurring intervals, either man, for some unknown reason, becomes more susceptible, or environment, again for an unknown reason, becomes favorable to its great increase and extension over the earth.

The man's physiognomy was interesting. Looked at from different angles, his face had the paralysis agitans or the exophthalmic appearance. I have seen this combination of two physiognomies in many cases.

CASE 4.—During childhood, this patient had frequent attacks of sore throat. He enlisted in the navy when 18 years old, and served for three years. Soon after enlistment, he was sent to a naval hospital because of persistent cough and loss of weight, with the tentative diagnosis of pulmonary tuberculosis, but after one month's treatment, he was discharged cured, and returned to duty. In August, 1920, he was discharged from the service "physically fit to be reenlisted." He then started to work as a pipe fitter. During the summer of 1923, when he was 23 years old, he was working in a damp cellar and developed sore throat, stiff neck and headache. He stayed away from work two days, but then felt well and so remained until six months later, when he noticed "a jerky" sensation in his legs while walking a short distance or standing a short time. After several months, he began to show festination. His own description of the trouble was: "When I start to walk I must go faster and faster and it is hard for me to stop short." Today he presents the typical picture of the paralysis agitans type of epidemic encephalitis.

His heart and lungs are normal. Mentally he is in good condition. The blood and spinal fluid Wassermann tests are negative.

The closest questioning revealed no acute illness immediately before his present trouble came on. He never had influenza. He admitted opportunity for venereal infection, but denied its ever having occurred.

CASE 5.—*History.*—The patient was 25 years old. Her father died of heart disease, and her mother had St. Vitus' dance when a child, and also died of heart disease. One sister died after childbirth, some months after the removal of a goiter; one sister was hysterical and had an epileptic child. The patient married at 18, left her husband after three months because of pus tube infection, married a second time at 19, and left the second husband because of cruel treatment. A family and personal history such as this in early maturity is a portent of evil to come.

In January, 1923, she became careless in appearance, and at first had a staring look and thick speech. She tired easily. Some months later, she began to have repetition in speech and would sit about the house doing nothing.

Examination.—Gait and station were those of paralysis agitans. The face was masklike. There were fine tremor of the tongue at rest and perseveration

in speech. She would answer a question in a few words and then repeat the answer many times. Consciousness was normal. She answered questions relevantly but was tearful and emotional. Her laughter was never uncontrolled. Her sister said that she had been very somnolent night and day for many months. Her height was 5 feet, her weight 113 pounds (51 kg.); she was small, well nourished and well formed. The right side of her face was smaller. Her expression was fixed and staring. The ears are poorly formed and asymmetrical. The blood pressure was: systolic, 100; diastolic, 70. The spine was not deformed, and percussion over it caused no pain. The left pupil was larger than the right, but both reacted well to light and in accommodation and there was no nystagmus. Tremor of the tongue on extension and of the eyelids when closed was present. There were constant sucking and chewing movements of the lips, which the nurse said continued during sleep. All the deep reflexes were present and active. There was an abortive patellar and ankle clonus. Station was good, and there was no dysarthria. The blood and spinal fluid Wassermann tests were negative.

Comment.—I have never seen perseveration in any other encephalitic patient, although a medical friend tells me that he has a patient showing it now, and I do not know how frequent it is. Notwithstanding the absolute denial by herself and her relatives of any acute illness, I believe that only epidemic encephalitis and influenza can produce such a disease picture, but that in this case, as in Case 1, there was also a congenital soil due to inherited or congenital causes.

COMMENT

These cases illustrate the fact that the poison of epidemic encephalitis may attack the central nervous system primarily and without any acute general symptoms of infection. The paralysis agitans type is, or seems to be, the most frequent. I have seen, in consultation, several cases of myoclonus, the picture of the so-called choreic type, in which there was no history of an acute febrile onset, but, although I have no doubt of the diagnosis, the histories are too fragmentary to be worth reporting at length.

A REVIEW OF SOME STUDIES OF DELINQUENTS AND DELINQUENCY*

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In a study of 4,000 cases of delinquency seen during some fifteen years, certain findings appear to be of value for shaping public policy in regard to the treatment of young offenders and the prevention of delinquency, and in demonstrating specific values in the professional approach to the understanding and treatment of misconduct. Two thousand of the cases were seen in Chicago, and comparisons have been made with two thousand seen in Boston. All cases are those of young, repeated offenders of an average age of from 14 to 15 when first seen; most of them are delinquents who have been in court more than once.

PHYSICAL CONDITIONS

Concerning general physical conditions, the 4,000 cases show no wide divergence from the general norms of the population, nor is there any significant difference between the two cities. Taking the age-weight findings as compared with general population norms, our cases cluster well about the normal curve of distribution. But there are one or two points of some significance: more boys show great overdevelopment than underdevelopment and no less than 70 per cent. of the girls are above the age-weight norm. This is in line with the common-sense observation that physical overdevelopment of girls has a tendency to be related to delinquency; and, of course, by far the most frequent delinquency of girls is concerned with sex matters.

Looking at the material more closely from a medical standpoint, one third of the subjects appeared to be in good physical condition, showing possibly only minor conditions obviously in need of attention. This is not far from the findings for the general run of young people. Since the beginnings of misconduct are so frequently found in early adolescence, the phenomena of puberty have some interest. Premature puberty occurs in 10 per cent. of the cases, whereas delayed puberty does not occur in more than 3 per cent. Sensory defects (vision, 15 per cent.; hearing, 2 per cent.) and diseased tonsils and adenoids (22 per cent.) are found in the proportions that one would naturally expect, certainly not with sufficient frequency to make them fairly regarded as marked causative factors in the production of delin-

* Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June, 1924.

quency. As an interesting example of what a special campaign can do, we may cite our findings on dental conditions. In Boston, a great community service is rendered by the Forsyth Dental Infirmary, which cooperates directly with the schools. The work of this infirmary and of other dental clinics shows the following results: In Boston, we found only 8 per cent. of patients with one or more badly carious teeth; in Chicago, there were 53 per cent.

Definite signs of organic nervous disease, exclusive of epilepsy, have been found in only 3 per cent. of the cases. Most of the cases of organic disease are cases of chorea, a disease that in some patients leads to most unfortunate behavior trends. Through special interest, I have looked carefully for early signs of multiple sclerosis; not more than four cases of the 4,000 could be fairly suspected of showing this disease. Even the earliest possible signs, such as absence of the abdominal reflex, are extremely rare.

Cropping up every now and again, particularly in neurologic circles, is the thesis that brain lesion, particularly from accident, is frequently the cause of delinquent behavior. In some case studies, which include in almost every case the developmental history, we have undoubtedly accurate information on this point. There can also be no doubt that in some remarkable cases a change of personality has directly followed a severe concussion, and that, although no statistics are available for a check group in the general population, there are a larger number of injuries of the head to be found among delinquents than among non-delinquents. Our total figures show that 3.5 per cent. of the patients suffered from injury of the head of some severity, each patient having suffered at least a period of unconsciousness.

The findings show no marked relation of congenital syphilis to delinquency in general. Even when one found somatic signs that earlier were interpreted as evidences of congenital syphilis, or when there was a significant family history, the Wassermann reaction was negative in nearly every case.

Concerning the older theory of the delinquent as a degenerate person, I may say that the ordinary so-called stigmas of degeneracy were not found in any great percentage of cases—6 per cent. of the total. If one rules out the mentally defective cases, small indeed is the figure for stigmas.

Although the geographic distribution of goiter is well known, specific findings are always of interest. For conditions presumably pathologic, namely, when there was present at least a small goiter, I found the following figures: 10.4 per cent. among the Chicago girls, 1.1 per cent. among the Chicago boys; 0.4 per cent. among the Boston girls, and none among the boys. These figures were for 1,000 cases in each city,

which had been carefully examined for the purpose. There is, therefore, the striking difference that young people in Chicago show goiter twenty-one times as frequently as those in Boston—the girls, ten times as frequently.

MENTAL CLASSIFICATION

As regards mental classification, there are no great general differences between the groups in Chicago and in Boston, except in certain particulars in which matters of special public service may have influenced the situation. In general, one can state that about 70 per cent. of the regular run of delinquents are normal mentally. By this I mean that by intelligence tests they are not defective and by other methods of examination they are neither psychotic nor psychopathic personalities. We found 13.5 per cent. of the total number clearly feebleminded. In addition, there were defective to some degree, but not certainly feebleminded, 9 per cent. It should be remembered that all of these cases represent particularly careful studies with regard to mental abilities.

The proportions with psychoses show a wide difference between Chicago and Boston; 5.6 per cent. in Chicago as against 1.1 per cent. in Boston. Perhaps this is partly accounted for by the part which the Psychopathic Hospital has played in the community in Boston, there having been for years a fine feeling of confidence in that institution, so that many patients go there voluntarily; the work of the outpatient department has also been extensive, and it has cooperated with various social agencies. Furthermore, we must not neglect the consideration that environmental conditions which make for lawlessness affect the mentally unstable first. Both the feebleminded and the psychotic tend to respond to unfortunate outer influences with much more ease and celerity than do the mentally normal.

We diagnosed about 3 per cent. as having psychopathic personalities. These are all instances in which the diagnosis was justified on grounds other than that of immediate misbehavior. I stand strongly against the use of this classification in the large way that frequently obtains, for several reasons, among them being the fact that in many cases in which such a diagnosis has been made elsewhere, the person with appropriate help has overcome his tendencies to misbehavior and then has shown no signs of instability. Another reason is that the causes of delinquency are numerous, and attempts at explanation by merely labeling the delinquent a psychopathic personality means most unjustifiable and unscientific simplification of the problem.

Constitutional inferiority in my opinion is a fairly concrete entity recognizable by rather definite criteria. About 2 per cent. of our cases fall in this category.

Epilepsy shows a remarkable difference in its incidence among Chicago and Boston delinquents—5.5 per cent. as against 1.8 per cent.

We may assume that a great cause for the difference is the fact that for many years in Massachusetts there have been special state institutions that care for epileptic children, so that there is a much smaller percentage of them in the community. The story of delinquency and crime among persons with uncontrolled epilepsy in Chicago is long, tragic and most expensive.

COMMENTS

Quite apart from the strictly medicopsychologic aspects of this study, we have discovered most extraordinary dissimilarities between these two American communities in regard to the types of offenses committed and the outcomes in reformation of criminality as the individual grows into adult life, variations which evidently cannot be explained by any such obvious factors as difference in nationalities or education, because these differences are not great. There seems, however, to be quite a noteworthy contrast between the methods of handling offenders through a much wider and wiser use of probation for older adolescents in Boston, and through much greater personal attention after parole from juvenile institutions. But perhaps even more striking in its effect on the prevention of delinquency is the much greater use in Boston of many social agencies, the social service departments of hospitals, the relation of settlements to neighborhood life, and so on. This is a matter for special professional interest, for, whether a mildly psychotic or psychopathic or neuropathic or defective person is going to be able to float morally in the community depends quite largely on the spirit of the community itself.

I have been greatly interested in following up persons who were both mentally abnormal and delinquent, in order to learn how they were handled and whether they succeeded or failed from the standpoint of conduct. I find that during the ten years or more, the period since first seen, comparatively few of the mentally abnormal received adequate treatment. For example, of a total of 284 abnormal repeated offenders in Chicago, fifty-six who were psychotic either were never committed to an institution for mental disease or did not remain in the institution; only fifteen so remained; seventy-five with psychopathic personalities were given no special treatment; only twenty-three defective persons remained in institutions for the feebleminded; 109 were never committed as mentally defective or did not remain in institutions for defectives. Some of these have in the long run been fairly successful, that is, as adults they have had no court records, and some of them have worked well and have had good family records. But the list of failures is, as might be expected, a long one, society paying dearly indeed for its neglect of proper treatment.

Taking 127 mentally abnormal boys recognizable as needing special care, ninety-three have been costly persons: at least six of them have

been murderers; sixty-four have been definitely criminalistic, many of them engaging in desperate affairs—there are no more desperate criminals than some of the feebleminded and psychopathic persons who have been allowed a long start in this direction—the others are the more occasional criminals or vagrants.

But I would not have it understood that I believe that even these abnormal persons are necessarily criminals. In another community the same types of persons do not become criminalistic to any such degree. Even among the mentally normal delinquents studied for after-careers we find just such records; of course, a greater proportion turning out favorably. Tracing 400 boys, I found thirteen murderers, seven of whom were normal mentally.

It is therefore clear that for the student of psychiatry the make-up and *mores* of society must have many implications both in regard to the genetics of conduct disorders, as one discovers by fair-minded case studies, and in regard to the after-care which is also affected by the habits of the social group and, perhaps more particularly, by the treatment afforded the delinquent himself.

DISCUSSION

Dr. S. Philip Goodhart, New York: So many important problems have been suggested by Dr. Healy's interesting paper that one can comment on one or two only. One very important question bears on the strictly intellectual processes of the individual and that part of his psyche which deals with the emotional reactions and their bearing on moral attitude and consequent behavior. In our weekly conference with Dr. Gregory, director of the psychopathic department of Bellevue Hospital in New York, where delinquents are often referred for medical opinion as to responsibility, we have made some pertinent observations. Among the cases observed in the ward and discussed at conference, were youthful delinquents whose conduct and type of crime suggested mental aberration even to the magistrates or investigators.

A striking feature of many of these cases was the evident discrepancy between the intellect generally and the reaction toward the moral turpitude. The condition in that respect suggests to me a striking identity with what we are seeing in cases of postencephalitis with conduct disorders. Just this moral deterioration with a strangely lacking emotional reaction and yet an apparent intellectual conception of the behavior as abnormal, is what seems to me to give character and type to the postencephalitic perverted conduct and to that often seen in the juvenile delinquent of familiar criminal type. In both the case of the organic—"acquired" pathologic case—and the perhaps "congenital" juvenile delinquent, there stands out in many that peculiar psychologic attitude toward the misbehavior: a want of critique, a superficial psychic reaction with defect in the depth of conscious appreciation. This want of emotional feeling, tone and real appreciation that comes from true understanding is perhaps in part responsible for a failure of will and maintained resolution necessary to inhibit abnormal impulse and desire. I wonder whether Dr. Healy has inquired into any cases of postencephalitis with conduct disorders?

Dr. William Healy: Our experience with cases of epidemic encephalitis is not very large, but the patients that we have seen show a lack of inhibitions similar to that of which Dr. Goodhart speaks. I think it resembles, however, the mental attitude of a good many delinquents in general, and I sometimes wonder, as I look about the world, whether, after all, this is so very different from much behavior that we see around us all the time; some delinquents have happened to adjust themselves to special forms of activity which the law proscribes; others may be just as uninhibited in social behavior, but in ways of which the law does not take cognizance.

THE MECHANISM OF PAPILLEDEMA*

WALTER R. PARKER, M.D.
DETROIT

In general, there are three principal theories advanced to account for the causation of choked disk: the inflammatory, the toxic or chemical and the mechanical. In addition to these theories, it has been suggested that various combinations of the factors involved in them may be regarded as the possible cause of the swelling of the disk in cases of increased intracranial pressure.

The mechanical theory of the causation of choked disk depends on increased intracranial pressure, increased intravaginal pressure, disturbance of the return blood flow and obstruction of lymph flow through the intravaginal to the subarachnoid spaces. Whether or not the increased intracranial pressure must be associated with increase in the amount of cerebrospinal fluid is still in dispute.

The original theory of von Graefe that the increase of intracranial pressure caused by a tumor led to compression of the cavernous sinus, and that this gave rise to an engorgement in the ophthalmic vein, was disproved by Sessman, who showed that the ophthalmic vein had such free connection with the facial that an obstruction to its flow into the sinus could not produce a choked disk.

The discovery of Schwalbe that the intravaginal space at the optic nerve communicates with the subarachnoid space of the brain led Schmidt-Rimpler to advance the theory that when there was an increase of the intracranial pressure a quantity of cerebrospinal fluid was pressed into the lymph spaces of the sheath of the optic nerve, leading to a papilledema and a secondary venous congestion.

Manz could not confirm the claim of Schmidt-Rimpler that a system of canals in the lamina cribrosa communicated with the arachnoid space, but added to the Schmidt-Rimpler theory the suggestion that by means of hydrops of the sheath, the intracranial pressure caused compression of the vessels in the end of the optic nerve as they pass through the lamina, which pressure resulted in an edematous swelling and infiltration. This so-called Schmidt-Manz theory is generally accepted by many as offering the most satisfactory explanation of choked disk. Cushing and Bordley believe that the changes are due, first, to mechanical, and secondly, to toxic or inflammatory causes. De Schweinitz says:

> In general terms, it is probable that choked disk is produced by a combination of factors. In this combination increased intra-cranial tension or pressure is most prominent, and the mechanical theory of its pathogenesis offers the most satisfactory explanation.

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*Read before the American Academy of Ophthalmology and Oto-Laryngology, Montreal, Canada, October, 1924.

As the results of the experimental work which I have conducted seem to confirm the mechanical theory, I shall limit my remarks to this phase of the subject and not attempt a complete review of the literature.

In general, the intracranial pressure is exerted equally in all directions, and if this pressure be sufficiently increased it manifests itself first along the paths of least resistance. The resistance at the disk to pressure from within would have as one element in it the tension of the eyeball. The globe showing the lower tension would offer the lesser resistance, and the nerve in that globe would, therefore, be the one first to show an edema at the disk if either nerve head were to become affected. Naturally, if the two globes had the same tension, both disks would be involved at the same time to an equal degree.

To test this theory, a series of experiments were conducted on dogs and monkeys in the following manner:

A sclerocorneal trephine operation was performed in one eye in order to make the two eyes different as regards the tension. After a marked difference in tension had been established, the intracranial pressure was increased by various means that need not be enumerated. It should be said, however, that in each instance the intracranial pressure was made on the side of the brain opposite the eye in which the tension had been reduced. The experiments were carried to a satisfactory conclusion in nine dogs and three monkeys. In every instance, the swelling of the disk appeared first in the eye showing the lesser tension. If the papilledema is not mechanical in origin, it is difficult to explain why the swelling of the nerve should always appear in the eye of lesser tension, especially when the eye first involved was invariably on the side opposite the location of the tumor.[1]

While the results of these experiments were most convincing, confirmatory clinical evidence that the onset of choked disk is in the eye of lesser tension, is not always so satisfactory. The uncertainty of taking accurate readings of the tension of the globe, the lack of opportunity for making an early observation in brain tumor cases, together with the great difficulty of differentiating the inflammatory from the edematous changes, and the early from the late changes in the nerve head, all contribute to the complication of the problem. It is essential, also, to remember that a choked disk is only an incident in the life history of a brain tumor; that is, the disk becomes edematous, swells to a certain maximum degree, and in time the swelling recedes and atrophy may follow. Again, an eye that may show an initial lower tension may later, in the presence of an edema of the nerve with hemorrhages, show a secondary glaucoma that will result in a reversal of the relative hardness of the two eyes.

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1. Parker, Walter R.: The Relation of Choked Disk to the Tension of the Eyeball, J. A. M. A. **67**:1053 (Oct. 7) 1916.

The difference in the tension of the two eyes is a determining influence in the onset of choked disk only so far as it indicates which eye will be the first affected. The eye first affected need not necessarily be the one in which the disk shows the greater swelling at the time of observation. When both disks are affected, the first examination may reveal an early edematous change in one eye, while the other may show signs of beginning atrophy. The disk in the softer eye may show an older process with less degree of swelling than that seen in the nerve involved later.

In one instance in which the pressure of a brain tumor was suspected and before any evidence of choked disk was present, I predicted that the papilledema would appear first in the eye that tested the lesser tension of 3 mm. of mercury. Later, I had an opportunity to observe the swelling appear first in the nerve of this eye. Dr. James Bordley has had a similar experience.

The difference in the tension between the two eyes need not be marked to be an influence on the onset of papilledema so long as the difference is constant. In the cases here reported, the minimum of difference as recorded on a Schiötz tonometer was 2 mm. of mercury.

Why the maximum degree of swelling at the disk in cases of intracranial pressure is limited to 7 or 8 diopters is not certain. It may possibly be explained by assuming that either the connective tissue in the laminae is put on the stretch with increasing resistance to the pressure from behind, or the edematous condition of the tissues at the site of the laminae acts as a stopper offering its own resistance. A combination of the two conditions might also be possible. Whatever the explanation may be, it is a well-known clinical fact that the swelling of the disk never exceeds about 3 mm. in height.

The clinical observation that cases of high myopia so rarely show a papilledema in the presence of increased intracranial pressure may be explained by assuming that in cases of posterior staphyloma the tissue composing the cribiform lattice is put on a tension, thus increasing the resistance at the papilla to the pressure from behind.

A clinical study of choked disk in its relation to the tension of the eyeball was made in twenty-three proved cases of brain tumor. The cases studied in this connection may be classified in groups as follows:

Group I. Cases showing greater degree of swelling in the eye of lesser tension .....No. 6

Group II. Cases showing an equal amount of swelling of the disk with the tension equal in the two eyes.....No. 10

Group III. Cases showing the greater amount of swelling in the eye of higher tension. Process older in the eye of lesser tension .....No. 2

Group IV. Cases showing the same amount of swelling in the two eyes but of different tension. Appearance of disk same in both eyes.....No. 5

In Group III the process in the disk of the softer eye was distinctly older than in the other eye, although the amount of swelling was the same in each. This difference in the appearance of the two eyes would at least suggest the possibility that the swelling appeared first in the eye of lesser tension.

The tension in the cases in Group IV was different in the two eyes, while the amount of swelling remained the same in amount and appearance. These cases are, therefore, classed as exceptions to the rule.

Assembling the results as tabulated, eighteen cases, or 78.2 per cent., conform to the rule, and five cases, or 21.7 per cent., are exceptions or unexplained.

As a result of the experimental work and clinical observation, there were 100 per cent. of the cases in the experimental work and 78 per cent. in the clinical cases observed bearing out the theory that choked disk in cases of increased intracranial pressure manifests itself first in the eye of lesser tension, or in cases in which the tension of the two eyes is the same, both disks become involved at the same time to an equal degree.

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THE USE OF LIPIODOL IN TUMOR OF THE SPINAL CORD

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In the practice of medicine we must look more or less askance at any new diagnostic procedure which may carry a considerable element of danger. For this reason it is wise to consider carefully the use in the cerebrospinal space of a substance opaque to the roentgen ray.

Lipiodol is a preparation of poppy seed oil in which iodin has been incorporated. The iodin is not simply in solution but is in the form of a fairly stable chemical combination. For best results the iodin content should be 0.45 gm. per cubic centimeter. The preparation should be clear and of a yellow or greenish-yellow color. Brown or turbid oil has deteriorated and should not be used. Lipiodol was originated by La Fay [1] four years ago and used by him in the treatment of epidemic encephalitis. Finding that this substance was not especially irritating to the meninges and was opaque to the roentgen ray, Sicard and Forestier [2] injected it by spinal puncture and demonstrated spinal cord compression. Feeling that such a procedure might well mark a distinct advance in the diagnosis of spinal cord tumors, Dr. J. B. Ayer and I obtained some lipiodol and used it first in animals and then in the human subject.

The data presented in this paper, with the exception of the animal experiments, have been gathered from twelve cases. Nine of these have come under my personal observation at the Massachusetts General Hospital, and the remaining three I am permitted to report, two through the kindness of Dr. J. B. Ayer and one through that of Dr. Donald Munro. The injection of lipiodol in my cases has been made by Dr. Ayer, Dr. Henry Viets and Dr. Hugo Mella. The roentgenography has been done largely by Dr. J. D. Camp, and Dr. Frank Fremont-Smith examined the spinal fluid.

In our earlier experimental work with lipiodol in cats, Dr. Ayer and I found marked cellular reaction in the cerebrospinal fluid, hence hesitated to use it in the human subject. We [3] briefly reported these

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1. La Fay, L.: Traitement de l'encephalite epidemique par les injections fortes d'huile iodee, Bull. general de therap., Paris **172**:163, 1921.

2. Sicard and Forestier: Methode radiographique d'exploration de la cavite epidurale par le lipiodol, Rev. neurol. 1264, 1921; Methode general d'exploration radiologique par l'huile iodee (lipiodol), Bull. et mém. Soc. méd. d. hôp. de Paris **46**:463, 1922.

3. Ayer and Mixter: Arch. Neurol. & Psychiat., abstr. **11**:499 (April) 1924.

findings. We also sought for some less toxic substance and for one that would move more freely and rapidly in the cerebrospinal space, but without success.

In our experimental animals we used from 1 to 2 c.c. of lipiodol, which would be equivalent to a dose of from 20 to 40 c.c. in man, with the result that one animal died and the others showed definite cellular reaction in the cerebrospinal fluid. As the animals that survived showed no permanent ill effects, we began cautiously to use the oil in patients, the dosage being from 1 to 2 c.c. Our first patient received injections into the lumbar sac, and was placed in the Trendelenberg position following injection. Roentgenograms clearly showed the oil, but it seemed to remain in irregular masses in the lumbar space and lower dorsal region. These earlier failures were probably due to insufficient lowering of the head of the patient. Later cases and animal experiments have shown that this oil may adhere to the meninges or nerve roots in droplets or even in large masses, causing the appearance of definite blocking of the cerebrospinal canal. This "false arrest," as it is called by the French, was striking in one animal. We supposed that a drop of mercury placed in the arachnoid space of a cat could be rolled around as one runs mercury around the bottom of a beaker. Much to our surprise we found that it was about as difficult to move in the cerebrospinal space as lipiodol. It was necessary to shake the cat much as one would a clinical thermometer. Even had the mercury moved freely, it would not have been available for the human subject owing to its toxicity. Since those earlier cases we have injected lipiodol into the cisterna magna in cases of suspected compression of the cord, reserving lumbar injection for the cauda equina. We have never attempted to outline both the upper and lower limits of the same tumor, feeling that to do so required a double dose of lipiodol and that of the two, the upper limit was the more important. Cervical and dorsal injection have not been used in this series, because we felt that the danger was disproportionate to the advantage.

The meningeal reaction in animals reached its height from twenty-four to forty-eight hours following injection when counts of 1,000 cells with moderate increase in the protein content of the cerebrospinal fluid were noted. The reaction subsided gradually, and by the tenth day the fluid was normal. In the one case in which we examined the spinal fluid on the day following injection of lipiodol we found a count of 900, mostly polymorphonuclears. This reaction had entirely subsided at the end of ten days. I also noted this meningeal reaction at operation in two patients operated on twenty-four and forty-eight hours, respectively, after the injection of lipiodol. In both patients the meninges appeared somewhat thickened and reddened, and the cerebrospinal fluid was

slightly turbid. De Martel [4] speaks of this inflammatory change in the meninges, although he does not mention the turbidity of the cerebrospinal fluid. All of our patients have had a moderate amount of headache and backache, which disappeared after a few hours or days, and a moderate rise in temperature. Lipiodol apparently remains in the canal almost indefinitely. We have shown it apparently unchanged in the lower end of the lumbar sac eight months after injection. In none of our cases have we noted any increase in paralysis, and we have had no fatalities.

I believe that such a substance should not be injected into the cerebrospinal space unless the possible dangers are more than outweighed by the more exact diagnosis to be obtained. For this reason, as time went on, we modified our technic. I think that there can be no true

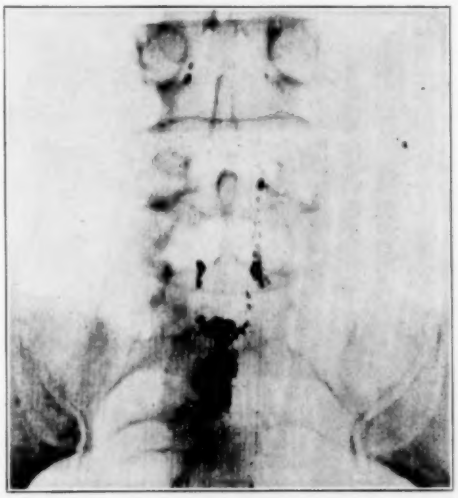

Fig. 1.—Lipiodol in the lumbar sac eight months after injection.

arrest of lipiodol in the subarachnoid space in the absence of spinal subarachnoid block as demonstrated by Ayer's technic, and for that reason I advise against its use unless block has first been demonstrated. Suspected lesions of the lower cauda equina is of course excepted. In order to fulfil these requirements and to permit all the punctures necessary to be performed at one sitting, we have adopted a definite routine which so far has seemed satisfactory. Sometimes these patients have had the presence of block demonstrated before their admission to the hospital. If so, the steps necessary to prove that fact are omitted.

The suspected level of cord compression is localized as closely as possible by neurologic examination and roentgenograms centering at the

4. De Martel, T.: Le traitement operatoire des tumeurs de la moelle et de ses enveloppes, Rev. neurol. **1**:701, 1923.

suspected level. Should these plates be positive, as for example in malignant disease of the vertebrae, the use of lipiodol would usually be unnecessary. If negative, the patient is prepared for combined cistern and lumbar puncture. Lumbar puncture is performed, pressure readings taken, jugular compression performed, and the fluid rapidly examined for protein, with the needle in situ. If there is no suggestion of block, nothing further is done. If block is proved or suspected, the cisterna magna is punctured, with the lumbar needle still in place. The presence or absence of block is confirmed; if block or partial block is present, the lumbar needle is withdrawn, and 1.5 c.c. of lipiodol is injected into the cisterna magna, care being taken that the oil is not broken up into globules in the syringe by water or air. The patient is then sent to the

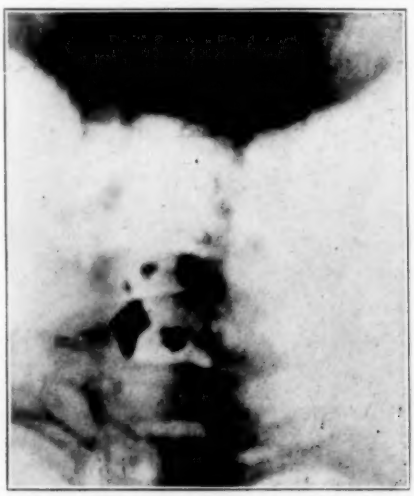

Fig. 2.—Cisternal injection. Note partial cap above a cervical neurofibroma.

roentgen ray room in the prone position; fluoroscopy is performed to locate the oil, and the patient is then put in the sitting position. In the future I hope to change this procedure by performing the injection in the roentgen-ray room with the patient in the inclined position as does Laplane.[5] Roentgenograms are taken at once and usually repeated in a few hours and again the next day. Occasionally plates are taken forty-eight hours after injection. Both anteroposterior and lateral views should be taken.

The interpretation of the films is of great importance. The lipiodol occasionally tends to move slowly and even to stop and pile up in the

5. Laplane, L.: Le radio-diagnostic des affections intra-rachidiennes par le lipiodol sous-arachnoidien. La forme pseudo-pottique des tumeurs intra-rachidiennes, Paris, Amedee Legrand, 1924.

canal for a short time. Such temporary arrest is of little or no significance, and seems to be due to adherence of the oil to the walls of the canal or to the nerve roots. In our experience rounded masses of oil sticking irregularly in the canal for a few hours are of no significance. The shape of the oil shadow, particularly its lower border, is important. A cap or crescent whether complete or incomplete has meant, in our few cases, extramedullary tumor or Pott's disease with intraspinal abscess, while narrow lines of lipiodol at either side with an irregular mass above suggest a fusiform enlargement of the cord itself.

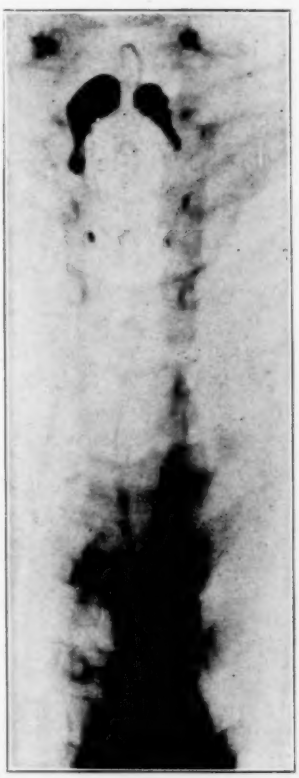

Fig. 3.—Cisternal injection arrest in upper dorsal region. Note cap above a neurofibroma. (Kindness of Dr. J. B. Ayer.)

We have injected lipiodol by lumbar puncture in two cases of suspected cauda equina tumor. The injection was satisfactory, but showed irregularities that we were unable to explain at that time. Exploration failed to reveal tumor. In my opinion, the error was due to lack of knowledge in reading the roentgenograms. After a little experience with positive cases, the method should be accurate and satisfactory. We have not intentionally used epidural injection, though in one patient we

accidentally obtained good extradural roentgenograms, probably on account of extravasation through the needle hole.

Partial review of the literature of this subject shows that following the publication of Sicard and Forestier's article, the use of lipiodol was taken up in France, but for some time not elsewhere. In the French literature are numerous reports of isolated cases and groups of cases and also a good deal of discussion from which valuable information may be gleaned. There are also a moderate number of reports to be found in the English, American and German literature. Recently Laplane,[5] working apparently in Professor Sicard's clinic, has assembled the French reports, grouped the cases and published a monograph, which

Fig. 4.—Cisternal injection; true arrest above an intraspinal abscess in the dorsal region.

is the first really authoritative statement on this subject. His deductions are based on a study of 300 cases, a far larger series than any one else has had the opportunity to observe.

In the whole series I can find no report of a fatality or of increase in paralysis following the use of lipiodol. Laplane states that the injection is usually painless, that there may be root pain in the vicinity of tumor and that the reaction in the cerebrospinal fluid is very mild. This opinion is also held by Sicard and Forestier,[6] and others. I have tried to remove the oil at operation, but without much success. It is possible that the

6. Sicard, Forestier and Laplane: Radio-diagnostic lipiodole au cours des compressions rachidiennes, Rev. neurol. **1**:676, 1923.

irritant action which Ayer and I have noted has been due to deterioration of our supply of lipiodol, though there is no evidence of it on inspection. Sicard, Haguenau and Laplane [7] warn us that it is important to allow six days to elapse after spinal puncture before lipiodol is injected. I think this point is well taken on account of the danger of cerebrospinal fluid leakage through the puncture in the dura with narrowing of the cerebrospinal space, but I do not feel that the removal of a small amount of fluid at the time of injection is an important factor. We have done that habitually in our studies and have seen no bad results.

I can find no mention of the proof of spinal block before injection of lipiodol, which we consider an important part of our technic, and I

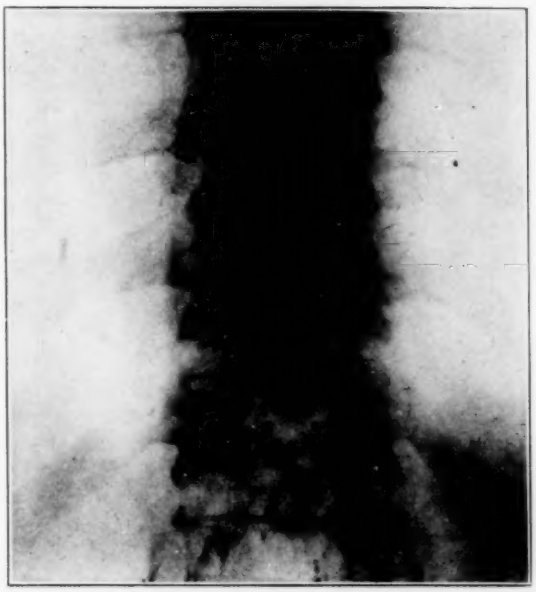

Fig. 5.—Lumbar injection; patient inverted; false arrest. Note absence of cap.

can find no mention of true arrest of lipiodol in the absence of block. Babinski [8] suggests a doubt as to the value of the procedure in all cases when he asks whether compression may occur even if normal descent of the oil is noted. Elsberg,[9] in a letter of recent date, suggests that in cases of tumor with absence of block this very thing would happen. There is no doubt that occasionally, with small tumors or those rare

7. Sicard, Haguenau and Laplane: Transit lipiodole rachidien; technique sous-arachnoidienne; resultats diagnostiques, Rev. neurol. **1**:1, 1924.

8. Babinski, J.: Sur l'epreuve du lipiodol comme moyen de diagnostic des compressions de la moelle, Rev. neurol. **1**:228, 1924.

9. Elsberg, Charles A.: Personal communication.

destructive new growths that cause narrowing of the cord, block may be absent. Such a condition has been observed three times by us.

Concerning the ultimate fate of lipiodol in the spinal canal little is known. There are no reports of late ill effects, but the photograph of a necropsy specimen obtained eleven months after injection published by Laplane [5] is sufficient to give much food for thought. Such a cyst involving the cauda equina might well cause symptoms.

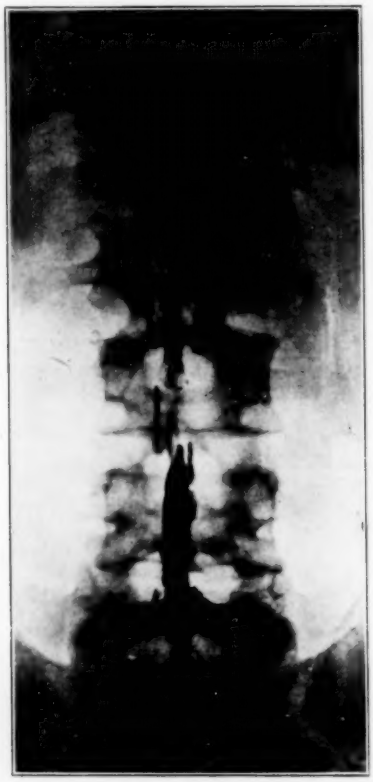

Fig. 6.—Lumbar injection; lipiodol in lumbar space. No tumor found at operation.

We find considerable evidence as to the accuracy of diagnosis by lipiodol. As well as those authors already mentioned, we have Sargent,[10] Ironside and Shapland,[11] Souques, Blamoutier and Massary,[12] Foix,[13]

10. Sargent, Percy: Radiographic Localization of Spinal Lesions by Sicard's Method, Brit. M. J. **2**:174, 1923.

11. Ironside and Shapland: A Case of Spinal Compression Localized Radiographically by Sicard's Method, Brit. M. J. **1**:149, 1924.

12. Souques, Blamoutier and Massary: Rev. neurol. **2**:443, 1923.

13. Foix, Charles: Discussion of spinal cord compression, Rev. neurol. **1**:616, 1923.

Russell,[14] Grant,[15] Naffziger,[16] Prusik and Volicer,[17] and others who agree that localization in their hands has been accurate and satisfactory. Vincent [18] suggests that epidural injection may be more delicate in extradural lesions when he cites a case of Pott's disease in which epidural lipiodol was arrested while that injected into the subrachnoid space went by.

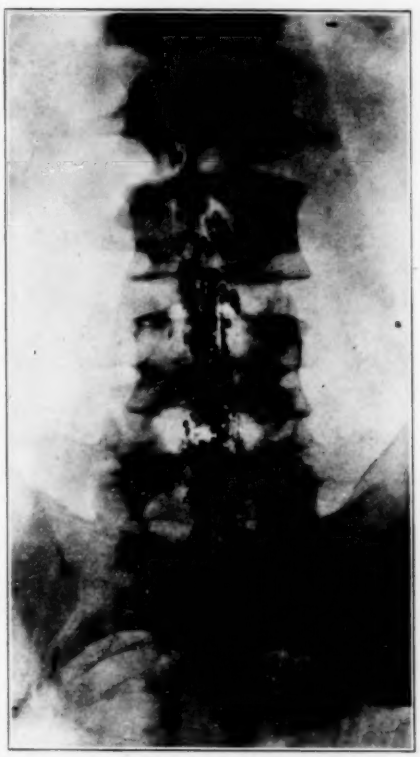

Fig. 7.—Same as Figure 6. Plates taken twenty-four hours later. Note that most of the lipiodol has leaked out of the spinal canal and is now present in the epidural space.

The interpretation of the roentgenograms is carefully studied by Laplane.[5] He feels that false arrest can be differentiated from true

14. Russell, Ethel C.: Localization of Spinal Block by Means of Iodized Oil, J. A. M. A. **82**:1775, 1924.

15. Grant, Francis C.: Clinic of Dr. C. H. Frazier, personal communication.

16. Naffziger, Howard: Personal communication.

17. Prusik and Volicer: Roentgen Examination After Intraspinal Injection of Iodized Oil, Casopis lekaru ceskych, Prague **63**:586, 1924.

18. Vincent, C.: Au sujet du diagnostic des tumeurs comprimants la moelle; de la valeur de la methode au lipiodol, Rev. neurol. **2**:562, 1923.

arrest by the shape of the shadow and by its fixation. In true arrest the lipiodol stops because it reaches an obstruction, and reversal of the position of the patient will cause the oil to flow backward away from the tumor. In false arrest the mass of oil has become fixed by adherence to the meninges and nerve roots and will not move on reversal of the patient.

How much reliance can be placed on lipiodol localization? De Martel [19] in the discussion speaks of four cases of arrest of lipiodol in patients operated on by him in whom no tumor was found and nothing to indicate why the lipiodol was arrested. He cites two of these as cases in which the lipiodol examination was the only real reason for operation and a third as one that had not had a careful neurologic examination. The question arises whether these patients would have shown spinal block if combined puncture had been used. De Martel feels, however, that one may localize spinal cord tumors exactly by the use of lipiodol, but only if already so diagnosed clinically. When there is disagreement between the clinical and lipiodol localization, the former is the more reliable. I cannot feel that the subject can be dismissed in this manner. There are too many factors involved and far too much variation in these factors. I should state it as follows:

Given evidence of block, a sharp neurologic level and an indefinite lipiodol level, one had better depend on the neurologic evidence.

Given a sharp localizing picture with lipiodol and a neurologic level, perhaps two or three segments below, one had better depend on the lipiodol level. This was the situation in one of my own cases, the tumor being found three segments above the neurologic level and accurately localized by lipiodol.

The results which we have obtained with lipiodol in conjunction with our previous knowledge of spinal subarachnoid block suggest the following conclusions:

We know that this oil is somewhat irritating to the meninges from its effects on the spinal fluid even though no immediate ill effects have been reported. We know also that the oil remains in the subarachnoid space almost indefinitely, where it acts as a foreign body. I feel strongly that a nonabsorbable substance of this character should not be used in the spinal subarachnoid space except when definite good is to be expected from its use.

If there is no evidence of block, the oil will find nothing within the canal to stop it, and injection will be useless. If accurate localization can be made by neurologic examination and the presence of block is demonstrated by puncture, it is useless again. We must remember, however, that exact localization is extremely difficult in some cases. Laminec-

19. De Martel, T.: Rev. neurol. **2**:444, 1923.

tomy is a severe operation, and if the exposure can be limited to three or four arches, the operative shock is less, as is also the resulting weakening of the spinal column.

Following this line of reasoning, I have concluded that I shall use lipiodol in all patients falling in the class in which block has been proved and in which the level is in the slightest doubt. We must accept lipiodol as a definite aid in the study of spinal cord compression, and must be ready and able to use it intelligently in a considerable percentage of our tumor suspects if we are to give them the benefit of exact localization.

By combining demonstration of spinal subarachnoid block and the careful use of lipiodol, we should be able to increase definitely our successful removal of cord tumors, at the same time being in a position to make a positive diagnosis of tumor and advise laminectomy earlier in the course of the disease. Owing to sharper localization, our operative mortality also should be less.

We must remember that the use of lipiodol is like the Wassermann test in syphilis, the basal metabolism in goiter and other new diagnostic tests. None is sure, and all of them will lead us astray some time if we neglect the fundamental basis of all diagnosis—physical examination of the patient.

LOCALIZED PROLIFERATIONS OF THE ARACHNOID

POSSIBLE RELATION TO THE ORIGIN OF TUMORS *

N. W. WINKELMAN, M.D.

AND

GEORGE WILSON, M.D.

PHILADELPHIA

In the routine study of many brains and cords, great variation in the arachnoid was noted. It was found that in certain cases small masses of cells were scattered irregularly within the arachnoid; the morphology of these cells proved them to be of arachnoid origin. The first description of these cell clusters was made in 1859 by Meyer [1] and in 1864 by Cleland,[2] although little attention has been paid to their findings. Cushing and Weed,[3] in 1915, were probably the first to corroborate Meyer's and Cleland's findings.

It would be well briefly to outline the present status of our knowledge of the normal structure of the arachnoid, with which we are here especially concerned. The arachnoid separates the subdural from the subarachnoid space. Histologically, it is composed of a very fine reticulum covered on both sides by its characteristic cells arranged so as to be only one layer in thickness. As Weed [4] definitely pointed out, there are trabeculae which project to and connect with the pia and which likewise are covered by arachnoid cells. These trabeculae divide the subarachnoid space into a weblike structure. "Except in certain definite areas, the arachnoid membrane on its outer surface is entirely separated from the inner surface of the dura mater." (Weed.[5])

As early as 1917, it was noted by Weed [5] that the morphology of the cells of the arachnoid depend apparently not only on their location, but also on their physiologic state. With the finding of localized changes in the arachnoid membrane in certain cases, it was felt that these might be dependent on conditions such as were discovered by Weed [5] and Essick [6] experimentally. These observers noted, for

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* From the Laboratory of Neuropathology of the Philadelphia General Hospital and from the Neurological Department of the School of Medicine of the University of Pennsylvania.

* Read at Fiftieth Annual Meeting of American Neurological Association, Philadelphia, June, 1924.

1. Meyer, L.: Virchows Arch. f. path. Anat. **17**:209, 1859.
2. Cleland: Glasgow M. J. **11**:148, 1863-1864.
3. Cushing, H., and Weed, L. H.: Bull. Johns Hopkins Hosp. **26**:367, 1915.
4. Weed, L. H.: Bull. Johns Hopkins Hosp. **31**:343, 1922.
5. Weed, L. H.: Anat. Rec. **12**:469, 1917.
6. Essick: Contributed to Embryology No. 42, Carnegie Instit. of Wash. Pub. No. 272, 1920, p. 377.

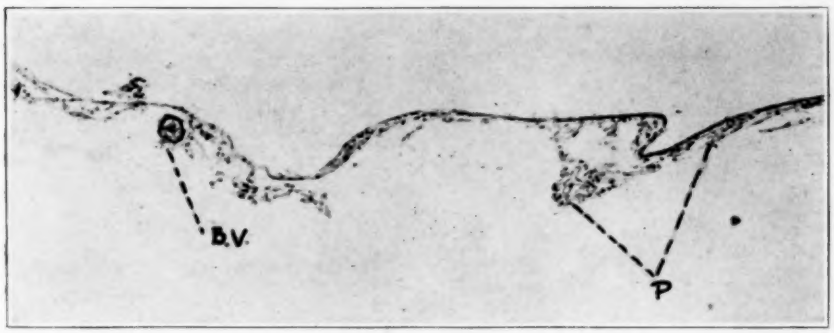

Fig. 1.—Normal arachnoid from a subject aged 45 (some of the pia [P] has remained attached with its blood vessels [B. V.]).

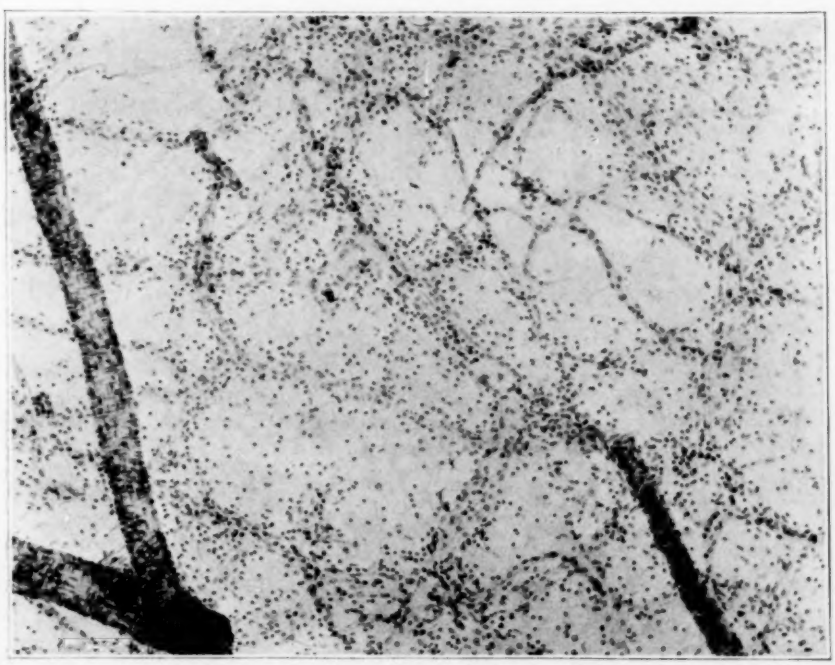

Fig. 2.—Arachnoid from a subject aged 6 months.

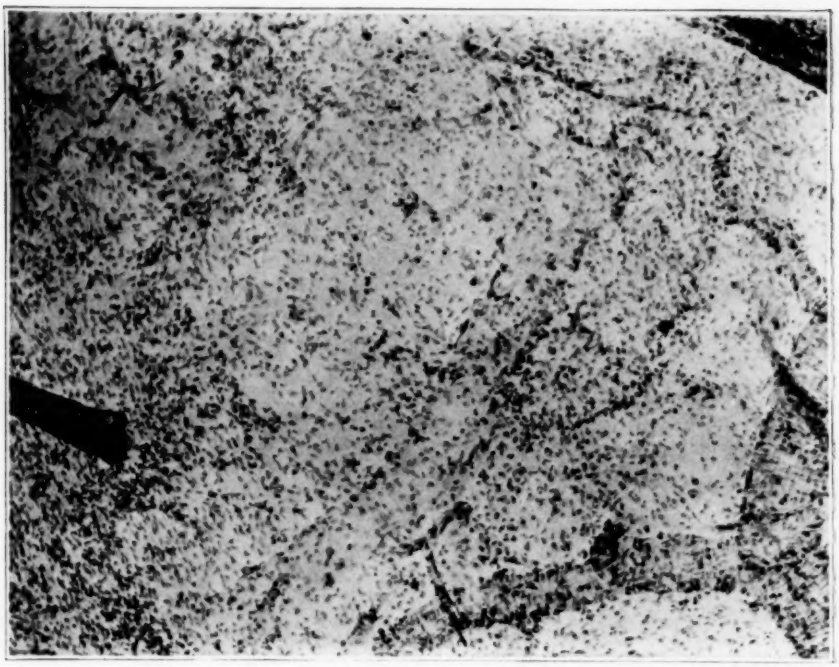

Fig. 3.—Arachnoid from a man, aged 24.

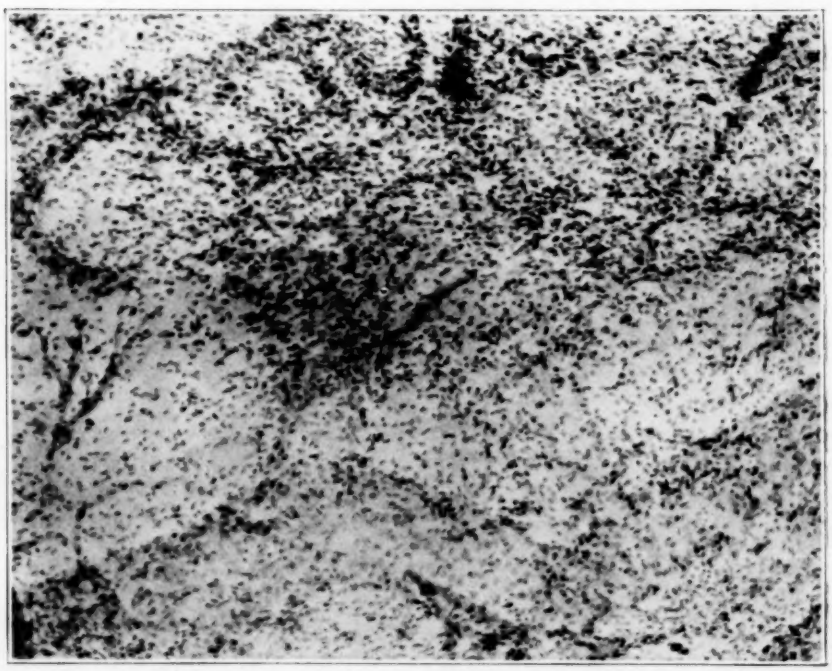

Fig. 4.—Arachnoid from a man, aged 36.

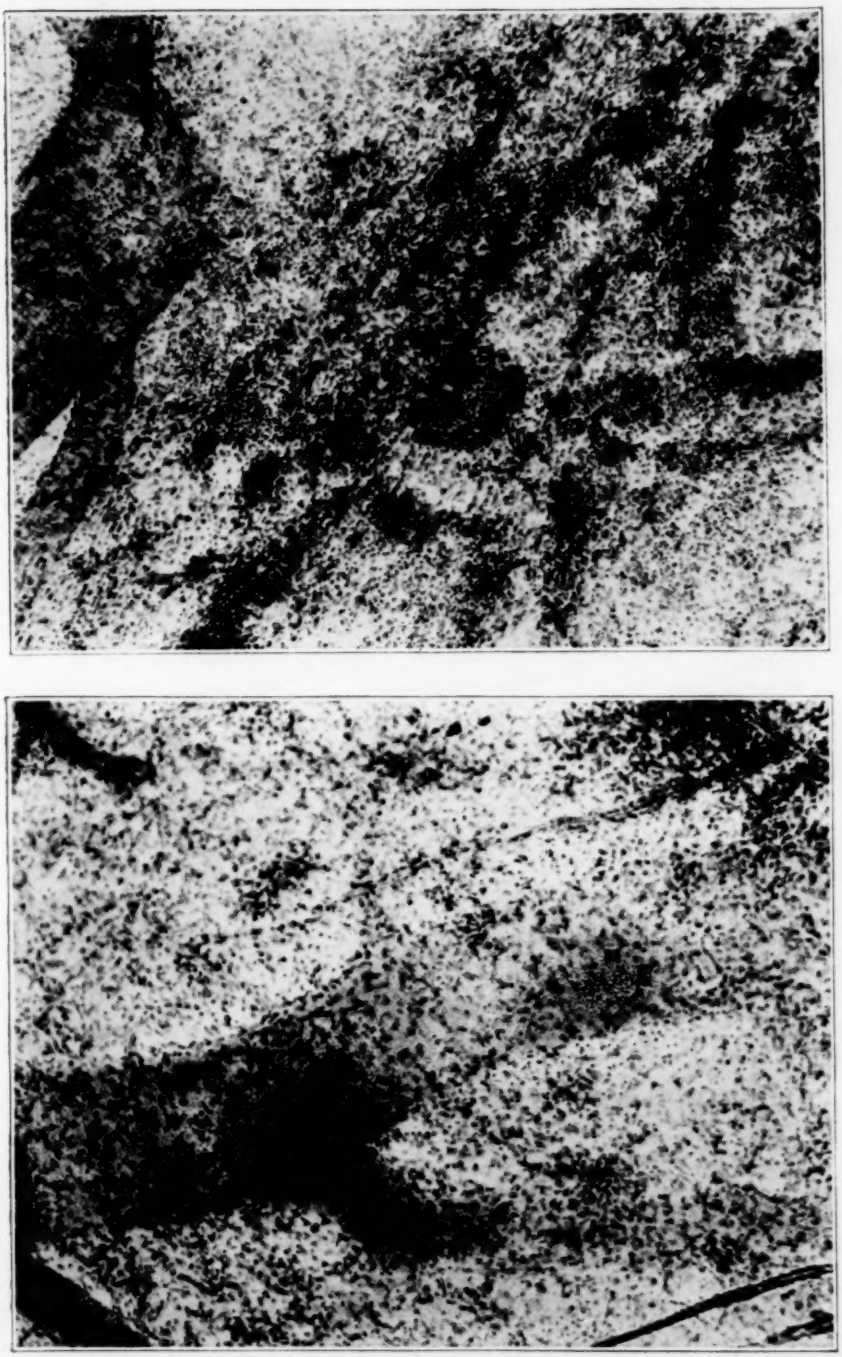

Fig. 5.—Arachnoid from a woman, aged 65.

Fig. 6.—Arachnoid from a man, aged 74.

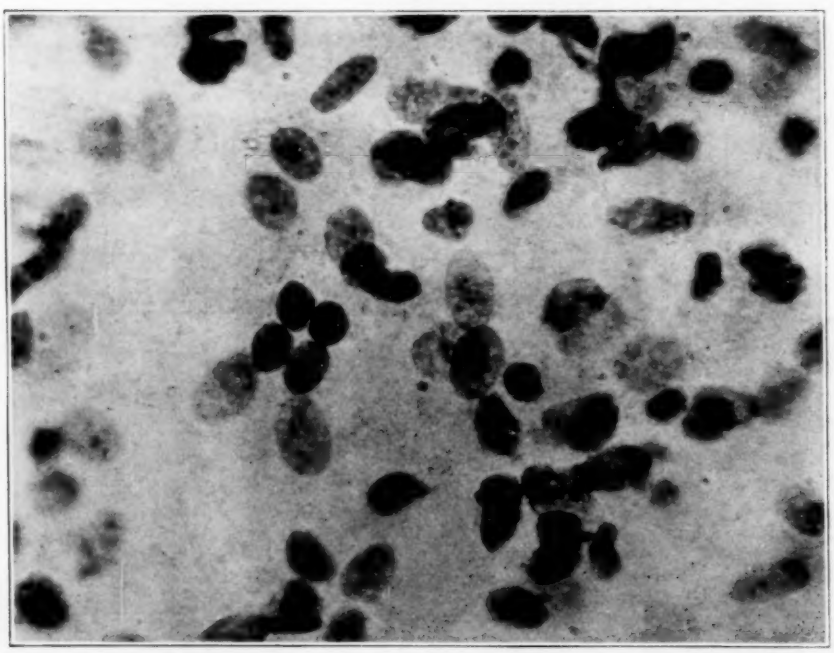

Fig. 7.—Oil immersion view of area from Figure 4 showing type of arachnoid cells.

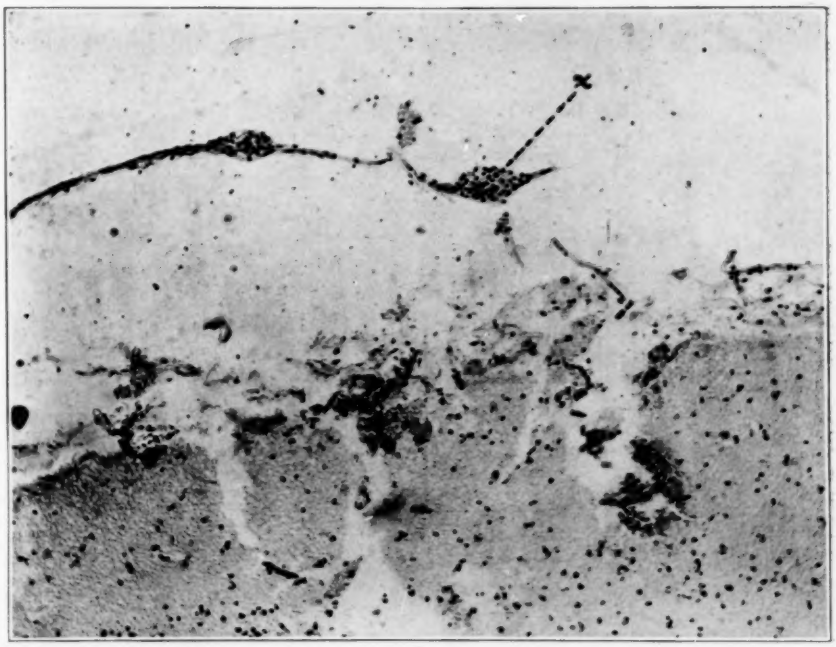

Fig. 8.—Cross sections of cell nests in arachnoid.

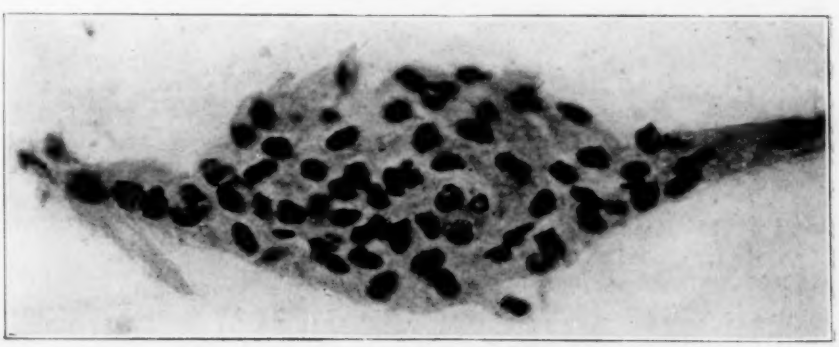

Fig. 9.—Oil immersion magnification of *X* in Figure 8.

example, that the injection of particulate matter into the subarachnoid space produced definite changes in the arachnoid cells. The same condition may occur in long standing infectious processes.

In order to determine the structure of the arachnoid at the various periods of life, a series of fifty brains was studied, taken from patients ranging in age from 6 months to 74 years. The method of study was

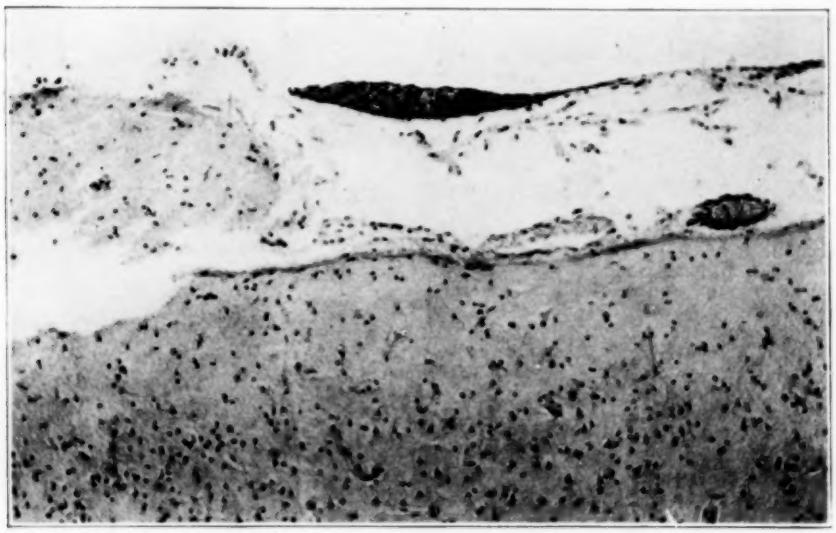

Fig. 10.—Flat endothelial proliferation.

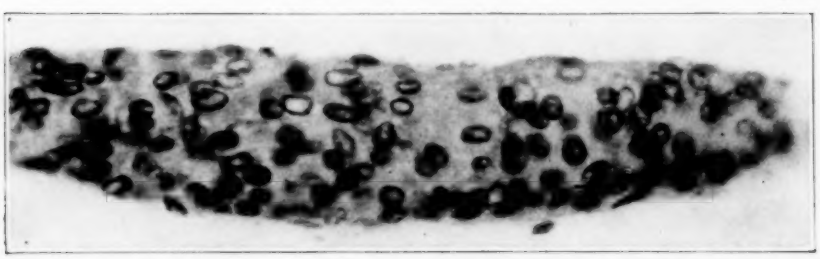

Fig. 11.—Oil immersion view of cluster in Figure 10.

as follows: The pia and arachnoid were dissected from the cortex in patches from 4 to 5 cm. in diameter. These were placed, pia down, on a slide which had been moistened with egg albumin, allowed to dry and stained with hematoxylin-eosin. In this way, large areas of the arachnoid could be studied rapidly and accurately.

The arachnoid from the youngest patient studied (6 months) (Fig. 2) showed a rather uniform distribution of cells without any localized

collections. (It is impossible to dissect off the pia from the arachnoid so that the pial vessels are seen shining through). As one studies the arachnoids in the succeeding decades of life, an increased number of cells is noted, as shown in Figures 3, 4 and 5. But one also notes a tendency to the formation of cell clusters, as observed especially in Figure 5.

But age itself is not the deciding factor in the production of these collections of cells, because in several instances, as for example Figure 6, an arachnoid comparable to that found at the age of 6 months was discovered in patients past 70. It is possible that stimulation of the cells to overgrowth may be secondary to their irritation in chronic infections or toxemias. Hassin's theory that all metabolized material of the brain is eventually poured into the subarachnoid space for elimination may have some bearing in this connection. Again, trauma must be taken into consideration in the production of these cell clusters. Weed,[4] in his experimental work on cats, found that their cell clusters appeared only in the older animals.

Cross sections of the cell nests (Figs. 8, 9, 10 and 11) show what might be called microscopic endothelial tumors. These collections are either flat or spindle shaped and correspond in a general way to the shape of full-grown endothelial tumors. The type of cell in these clusters is the same as that found in the normal arachnoid and in the so-called endothelioma.

SUMMARY AND CONCLUSIONS

A tendency to localized arachnoid proliferation occurs in people past middle life. Trauma and infection are important contributing factors or may even be the exciting causes. Endothelial tumors may take their origin from these cell clusters.

DISCUSSION

DR. M. ALLEN STARR, New York: It occurs to me that possibly these variations might be like the variations we find in the skin of different persons. May not the person with a coarse skin have a coarse arachnoid and the person with a thin skin a thin arachnoid?

DR. WILLIAM G. SPILLER, Philadelphia: In the *Journal of Medical Research* for August, 1903, I reported a case of lead encephalopathy with such collections of cells in the cerebral pia as those described by Dr. Winkelman.

THE CEREBROSPINAL FLUID IN TUMOR OF THE BRAIN *

R. G. SPURLING, M.D., AND C. L. MADDOCK, PH.D.
BOSTON

The value of a systematic study of the cerebrospinal fluid from a diagnostic standpoint is established beyond doubt in the fields of neurosyphilis and the acute inflammatory processes. Many consider it helpful in a few of the chronic degenerations of the central nervous system, most notably in multiple sclerosis. There have been but few extensive investigations carried on to ascertain the changes, if any, in the cerebrospinal fluid in tumor of the brain. Perhaps this is to be accounted for by the fact that lumbar puncture is known to be a dangerous procedure in advanced cases with foraminal herniation.

Of the observations made on the cerebrospinal fluid in tumor of the brain, those of Lange [1] are significant. In 1912 this author studied the colloidal gold reaction in five spinal fluids obtained in cases of brain tumor. He said that tuberculous meningitis, brain tumor, and hemorrhages into the subarachnoid spaces or ventricles all produced a spinal fluid showing the same gold curve. In a more recent contribution, from a series of sixty cases, Lange [2] seeks to establish a definite picture of the condition in spinal fluids in cases of brain tumor. This picture includes the following essentials:

1. A typical gold curve with color changes occurring in the dilutions above 1:80.

2. A moderate increase in total protein values. (Normal value = 20 mg. per 100 c.c. spinal fluid; average brain tumor value = 40 mg. per 100 c.c. spinal fluid.)

3. The almost invariable yellow coloration of the spinal fluid. He also states that the picture is diagnostic of brain tumor only when old hemorrhages due to trauma, apoplexy, operation or puncture are excluded, for these old hemorrhages give a picture identical with that obtained in brain tumor.

Moersch [3] reviews a series of 127 proved cases of brain tumor, out of which he selects nineteen cases in which there were unusual findings.

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* From the Surgical Service, Peter Bent Brigham Hospital, Boston.

* Read at a meeting of the Association for Research in Nervous and Mental Diseases, Dec. 30, 1924.

1. Lange, C.: Ueber die Ausflockung von Gold Sol durch Liquor cerebrospinalis, Berl. klin. Wchnschr. **49**:897, 1912.

2. Lange, C.: Was leistet die reine Liquor Diagnostik bei der Diagnose des Hirntumors? Mitt. a. d. Grenzgeb. d. Med. u. Chir. **33**:582, 1921.

3. Moersch, F. P.: Serology in Brain Tumors, J. Nerv. & Ment. Dis. **58**:16, 1923.

The spinal fluids were analyzed from the standpoint of cell counts, Lange's gold curve, the Nonne test and the Wassermann reaction. He found a high cell count in all of those nineteen cases, (8-679 cells per cubic millimeter, with a count most commonly between 14-20). Of the whole series, forty-one fluids were negative. He does not state what the picture was in the remaining sixty-seven cases.

Mouriz [4] concludes from the analysis of spinal fluids from eight cases of brain tumor that the most important points in the spinal fluid findings are an increase in total protein, with a low cell count and a characteristic gold curve. He also states that xanthochromia when present is a most important sign.

About one year prior to the present investigation, a series of cerebrospinal fluids, chiefly obtained by ventricular puncture during the course of operation from cases of suspected brain tumor in Dr. Cushing's clinic were examined by Dr. B. J. Alpers. The results obtained were sufficiently suggestive to warrant a continuation of the investigation. Our study was therefore undertaken with the hope of establishing a sufficiently definite picture in spinal lumbar and ventricular fluids from cases of tumors of the central nervous system to be of service in arriving at a diagnosis. An attempt was made to diagnose each case from the picture presented by the cerebrospinal fluid itself. This diagnosis was then checked up with the clinical evidence, and with operative and necropsy findings when such were available. In the final analysis of the data presented, certain uniform standards were established. It is not to be supposed, however, that the standards are fixed, for as additional data are accumulated, they will, no doubt, be modified to fit new facts. Emphasis is therefore to be put on the preliminary nature of this report in which the exact pathologic nature and position of the lesions, whether subcortical, submeningeal, or subependymal is not taken into consideration.

It should be mentioned that the fluids in cases of brain tumor have been secured only as a result of cautious preoperative tests, such as in the making of ventriculograms. Most fluids have been secured during the course of operative procedures. The predominant number of ventricular fluids is due to the fact that ventricular puncture is a routine step in the course of most operations for subtentorial lesions.

METHODS

The cerebrospinal fluids were examined in accordance with the following scheme:

General Appearance and Physical Characteristics.—Here clarity, colorlessness, turbidity of varying degrees, due either to cellular elements or bacterial contami-

4. Mouriz, J.: El Liquido cefado- Raquideo en Las Neoplasias Del Sistema Nervioso, Arch. de. neurobiol. **4**:13, 1924.

nation, and xanthochromia were noted. In order to quantitate the degree of xanthochromia present, a modification of the Bernheim icterus index[5] method was used. A series of tubes, containing solutions of known concentrations of potassium dichromate, ranging from a dilution of 1:100 to 1:200,000 was employed. The xanthochromic fluid was then matched in a comparator against the colors of the standards, and the xanthochromia designated numerically. As in the Bernheim scale the 1:10,000 potassium dichromate solution was regarded as unity. The number of any other dilution was obtained by making use of this simple mathematical expedient:

$$1 \times 10{,}000 = \text{No. of dilution} \times \text{dilution figure}$$

To illustrate:

$$1 \times 10{,}000 = 100 \times x$$
$$x = 100$$

Hence a dilution of 1:100 potassium dichromate solution would be placed at 100 on the scale. The xanthochromic index in the cerebrospinal fluid in the cases studied rarely exceeded one. Xanthochromic indexes are hence usually expressed by fractional numbers.

Cellular Content.—A determination of the number of red blood corpuscles was made on each fluid examined, even though there was no macroscopic evidence of contamination with blood.

White cell and differential counts were made following the technic of Levinson.[6] The diluting fluid was made according to the following formula:

Methyl violet	0.1 gm.
Glacial acetic acid	2.5 c.c.
Distilled water	50.0 c.c.

An ordinary white counting pipet was used. The diluting fluid was drawn up to Figure 1, and the pipet filled with spinal fluid to Figure 11, shaken thoroughly and allowed to stand for ten minutes to insure proper staining of the white cells and hemolysis of the red cells. A drop of the mixture was placed on a Levy counting chamber (Neubauer ruling) and a determination of the number of white cells made by taking into account the dilution of the fluid and the correction for the counting chamber. With this stain the nuclei of the white cells stand out clearly, thus making it quite easy to make a differential count in the fresh specimen. In using a staining fluid such as this, it is desirable to place in a separate chamber the sample of fluid to be counted, else the whole supply may become discolored with the dye, thereby obscuring the presence of xanthochromia. The customary procedure was to place about 1 c.c. of the fluid in a small test tube and from this to make a red and white cell count, using the remainder for a qualitative globulin determination. Thus the original sample arrived at the chemical laboratory uncontaminated.

Globulin: Qualitative and Quantitative.—These determinations were made according to the Nonne-Apelt,[7] reaction (Phase 1). The results were consistently read against a black background after the lapse of one minute. They were interpreted as follows: indistinct ring ±, distinct ring +, heavy ring + +.

An attempt was made in the earlier part of the problem to establish a quantitative method for the estimation of the globulin fraction of the protein

5. Bernheim, Alice R.: The Icterus Index, J. A. M. A. **82**:291 (Jan. 26) 1924.
6. Levinson, Abraham: Cerebrospinal Fluid in Health and Disease, St. Louis: C. V. Mosby Company, 1919, p. 226.
7. Nonne, M.: München. med. Wchnschr. **54**:2117, 1907.

content of the cerebrospinal fluid. A nephelometric method given by Pfeiffer, Kober and Field[8] was used, but was abandoned because of its decided limitations in ventricular fluids containing minimal amounts of protein, and because of its usage of large quantities of fluid. The results obtained in the few fluids examined pointed to a rather constant 1:3 ratio between the globulin and total protein, thus indicating either that a quantitative estimation of the globulin fraction is of no advantage over a quantitative estimation of the total protein value, or that the method lacked in accuracy and delicacy.

Total Protein.—Emphasis was placed on the estimation of the total protein in the cerebrospinal fluid. A modification of the Denis and Ayer[9] method was used. To 1 c.c. of spinal fluid, or 2 c.c. of ventricular fluid, an equal quantity of 23 per cent. sulpho-salicylic acid is added, and the mixtures shaken. The spinal fluid is further diluted by the addition of 10 c.c. of distilled water. The clouds thus produced are read in the nephelometer against a protein standard (45 mg. per 100 c.c. of fluid) which is precipitated and diluted in the same way as the spinal fluid. Since the relationship of

$$\frac{\text{Reading of known}}{\text{Reading of unknown}} = \frac{\text{Protein concentration of known}}{\text{Protein concentration of unknown}}$$

obtains only within narrow limits, it was found necessary to correct the calculated values on the basis of a curve obtained experimentally by plotting actual protein concentrations against nephelometer readings. The variations from calculated values, in the more concentrated protein solutions (i. e., 110 mg. and above) reached a value of 30 per cent. and over of these calculated values. Even with such a safeguard, we found it advisable to discard results that gave readings below 13 or 14 when the standard was set at 20. Two methods were used for bringing fluids containing high protein values within the limits assigned, the one, as indicated by Denis and Ayer in their original paper, diluting the spinal fluid with water previous to precipitation, the other diluting the fluid with water after precipitation, and thus exceeding in amount the allotted 10 c.c.

Lange's Colloidal Gold Curve.—This test was made on every fluid examined. The gold reagent was prepared according to Stitt's newest method,[10] which differs from previous methods in its omission of oxalic acid, and its use of a redistilled liquor formaldehydi. Freshly distilled water (once distilled and occasionally double distilled) was used instead of the traditional triple distilled. The reagent was standardized against fluids known to give one of the following characteristic reactions: (1) the negative fluid, (2) the fluid with the strong paretic curve, (3) the fluid with a well defined syphilitic curve.

Curves obtained were placed in the following categories (the classification adopted by this hospital):

1. Type I, the syphilitic curve, interpreted as positive or doubtful (1 2 2 3 1 - - - - -).
2. Type II, the paretic curve interpreted as positive or doubtful (5 5 5 5 5 5 4 3 1 -).
3. Type III, the curve characteristic of brain tumor and tuberculous meningitis, interpreted as positive or doubtful (- - - - 1 2 3 1 - -).

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8. Pfeiffer, Kober and Field: Sec. Exper. Biol. & Med. **12**:153, 1914-15.

9. Denis, W., and Ayer, J. B.: A Method for the Quantitative Determination of Protein in Cerebrospinal Fluid, Arch. Int. Med. **26**:436 (Oct.) 1920.

10. Stitt, E. R.: Practical Bacteriology, Blood Work, and Animal Parasitology, Philadelphia, P. Blakiston's Son & Company, 1911.

4. Type IV, the curve characteristic of meningitis other than tuberculous, interpreted as positive or doubtful (-----12334).

The Wassermann Reaction.—A routine test was made on each fluid. Dilutions varying in amount from 2 c.c. to 0.1 c.c. were employed.

The Sugar Content.—Sugar determinations were made in a routine manner on every fluid examined. The method of Folin and Wu[11] was followed. Use was made of the protein free spinal fluid filtrate, rather than of the spinal fluid itself (Alpers, Campbell and Prentiss[12]), since the material on hand included fluids with so wide a variation in protein content. A dextrose standard equivalent to 75 mg. per 100 c.c. of fluid was found most serviceable.

NORMAL CEREBROSPINAL FLUIDS

In order to establish a norm for the laboratory findings of fluids in cases of tumor of the central nervous system, it is first necessary to define the cellular, chemical and serologic relationships obtaining in normal cerebrospinal fluids. It is scarcely necessary to state that the normal fluid, particularly the ventricular fluid, is rarely found in our laboratories. Hence the laboratory worker finds it imperative to fix some more or less arbitrary standard for a basis of comparison. The normal values cited below were derived by averaging the results obtained from three distinct groups. Group 1 included proved negative cases which were negative serologically. (The term "negative serologically" as used in this paper means negative from the standpoint of all tests listed under the methods given. All fluids insufficient in amount to make analysis possible were discarded in the fixing of the normal standards for spinal and ventricular fluids.) Group 2 included proved cases of arachnoiditis that were negative serologically. Group 3 included unproved cases negative serologically. In ventricular fluids an additional group, Group 4, composed of proved brain tumor cases, negative serologically, was used.

The normal spinal fluid, as established in the manner outlined above, showed the following characteristics:

1. It was clear and colorless.
2. The white cell count varied from 0 to 8 cells, with an average count of 2 to 3 cells. These cells were mononuclears.
3. The globulin was negative by Phase 1 of the Nonne-Apelt reaction.
4. The total protein value varied from 21 to 47 mg. per hundred cubic centimeters of fluid, with an average value of 37 mg.
5. The gold curve was negative.

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11. Folin, O., and Wu, H.: J. Biol. Chem. **41**:3, 1920.
12. Alpers, B. J.: Campbell, C. J., and Prentiss, A. M.: The Spinal Fluid Sugar, Arch. Neurol. & Psychiat. **2**:653 (June) 1924.

6. The sugar content varied from 57 to 84 mg. per hundred cubic centimeters of fluid, with an average of 74 mg.

7. The Wassermann reaction was negative.

The normal ventricular fluid gave a picture that varied markedly only in one respect from that presented by the normal spinal fluid. This was in its protein value, which ranged from 5 to 18 mg. per hundred cubic centimeters, and averaged 7.8 mg. The sugar content showed more constancy in quality, 61 mg. per hundred cubic centimeters being a low normal, 79 mg. a high normal and 73 mg. the average value. From a large number of spinal fluids of medical cases submitted to the laboratory, on which sugar values were obtained, but which could not be classed as serologically negative because of insufficient data, the information was gained that the average sugar value of the spinal fluid was somewhat lower than that of the ventricular fluid, and that this average value was 69 mg. per hundred cubic centimeters of fluid.

THE FLUIDS IN CASES OF TUMOR: SPINAL AND CEREBRAL

The findings in spinal fluids varied markedly, depending on the location of the tumor.

Spinal Cord Tumor with Block.—All but one of the cases studied presented the typical Froin syndrome, hence were associated with block. The spinal fluid under these conditions presented the following characteristics:

1. It was xanthochromic and frequently clotted on standing.
2. The cell count was low, usually less than 3.
3. The globulin was markedly positive.
4. The total protein value averaged 3,600 mg. per hundred cubic centimeters of fluid.
5. The gold curve varied with the amount of protein present. Generally speaking, a total protein value below 1,000 gave a positive Type III curve, between 1,000 and 2,000 a doubtful Type IV curve, and above 2,000 a positive Type IV curve. In passing, it may be said that the type of gold curve produced seems to be dependent on the quantity of protein present.
6. The sugar value showed no significant changes.
7. The Wassermann test was negative.

Spinal Cord Tumor Without Block.—Here the findings were intermediate between those of spinal cord tumors with block and brain tumor.

Spinal Fluid in Brain Tumor.—The fluid here presented the following characteristics: (1) It was usually clear and colorless, slightly xanthochromic; (2) the cell count was low, usually less than 3; (3) the

globulin was positive; (4) the total protein value most frequently lay between 250 and 300 mg. per hundred cubic centimeters; (5) the gold curve fell in the Type III category; (6) the sugar content did not vary appreciably; (7) the Wassermann test was negative.

Ventricular Fluid in Brain Tumors.—In this fluid the variation was in the same direction as in the spinal cases, that is, in elevation of total protein, with the type of gold curve corresponding to that elevation. These fluids showed a normal protein content, that is, from one-third to one-fourth that of spinal fluids. This fact markedly influences the abnormal picture obtained.

TABLE 1.—*Findings in Spinal and Ventricular Fluids*

	Physical Characteristics	Cell Count	Globulin (Qualitative)	Total Protein		Gold Curve	Wassermann Test	Sugar
				Av.	Var.			
Spinal Fluids								
Spinal cord tumor (with block) 3 cases	Xanthochromic, clot forming	Less than 3	++	3,007	683 to 7,613	Positive Type III Doubtful Type V Positive Type IV	—	No variations
Spinal cord tumor (without block) 1 case	Clear and colorless	Less than 3	++	447	447	Positive Type III	—	No variations
Brain tumor..... 5 cases	1 xanthochromic, 4 clear and colorless	Less than 3	++ + +	291	154 to 469	Type III	—	No variations
Chronic arachnoiditis, 5 cases	Clear and colorless	9	—	35	..	Negative	—	No variations
Ventricular Fluids								
Brain tumor..... 27 cases	4 slightly xanthochromic	Less than 3	±	52	23 to 132	Type I and occasionally Type II	Neg. except 4 cases when positive in 2 c.c.	No variations
Chronic arachnoiditis, 8 cases	Clear and colorless	6	—	10	5 to 14	Negative	—	No variations

A statement of the conditions in ventricular fluids positive for brain tumor would include the following items:

1. The fluid was clear and colorless, rarely showing xanthochromia.
2. As in the spinal fluid, the cell count was generally less than 3.
3. The globulin was usually doubtfully positive.
4. The total protein value in the cases studied varied from 23 to 132, with an average of 52 mg. per hundred cubic centimeters of fluid.
5. The gold curve fell in the Type I group, occasionally approaching the Type II group.
6. The sugar content did not vary significantly.
7. The Wassermann test was negative.

PRESENTATION OF DATA

Fluids from 108 patients were examined. The diagnoses in sixty-five cases were verified either by operation or by necropsy, and in four cases by well established and generally accepted laboratory methods. These four cases include three of cerebrospinal syphilis and one of tuberculous meningitis; in the latter case the spinal fluid gave a positive inoculation test for the tubercle bacillus.

All bloody fluids were discarded. One fluid in the series showed a red cell count of 700; the rest were either negative for red cells or gave counts below 85. As to the justification for including the fluid with the erythrocyte content of 700, it may be said in passing that

TABLE 2.—*Analysis of Proved Cases*

	Number of Cases with Percentages									
	Spinal Fluids				Ventricular Fluids				Fluids from Subarachnoid Accumulations	
	No. of Cases	Correct	Incorrect	Questionable	No. of Cases	Correct	Incorrect	Questionable	No. of Cases	Correct
Tumor	12	9 (75%)	1 (8%)	2 (17%)	27	13 (48%)	7 (26%)	7 (26%)	..	..
Negative exploration or negative cases	6	6 (100%)	..	..	1	1 (100%)	..	..	..	..
Chronic arachnoiditis	5	5 (100%)	..	..	8	6 (75%)	2 (25%)	..	2	2 (100%)
Acute inflammatory processes (tuberculous meningitis)	2	2 (100%)	..	..	2	1 (50%)	1 (50%)	..	..	..
Central nervous system syphilis	3	3 (100%)	..	..	..	..	..	..	..	..
Total	28	25 (89%)	1 (4%)	2 (7%)	38	21 (55%)	10 (26%)	7 (19%)	2	2 (100%)

Grand total, 68 cases; correct, 48 (71%); incorrect, 11 (16%); questionable, 9 (13%).

work done in this laboratory on the effect of the red cell content on the total protein value and on the gold curve would indicate that only when red cells exceed 2,000 cells per cubic millimeter is a demonstrable change produced.[13] Fluids were excluded when information on either a cell count or a sugar determination had not been made, since one or the other of these was regarded as necessary for distinguishing between fluids presenting the picture of inflammation and those presenting the picture of tumor.

Table 2 includes the data on the sixty-eight proved cases in which an attempt was made to determine the diagnosis on serologic data alone. Of these forty-eight (71 per cent.) were diagnosed correctly, eleven incorrectly (16 per cent.), and nine were listed as questionable (13 per

13. Maddock, C. L., and Maddock, S. J.: Unpublished observations.

cent.). The questionable group includes those cases in which some portion of the data showed a variation from what was considered to be the typical picture.

There were in all thirty-nine proved cases of tumors of the central nervous system. From this group fluids were obtained from the lumbar region in twelve instances, and from the ventricles in twenty-seven instances. Of the twelve spinal fluids examined, the location of the lesions was distributed as follows: spinal, 4; supratentorial, 4; subtentorial, 3; suprasella, 1. Of the twenty-seven ventricular fluids examined, the lesions were found to be: subtentorial, 17; supratentorial, 7; suprasellar, 3.

Correct serologic diagnoses of tumor were made in 75 per cent. of the spinal fluids examined, incorrect diagnoses in 8 per cent., and 17 per cent. were listed as questionable. Of the twenty-four ventricular fluids examined, in 48 per cent. the lesions were diagnosed correctly, in 26 per cent. incorrectly and in 26 per cent. the diagnoses were questionable.

In analyzing the incorrect diagnoses we find the one mistake made in the spinal fluid group occurred in a case of cerebellar glioma. The seven errors made in the ventricular fluid group occurred in tumors of the posterior fossa. This consistency is striking. One cannot help wondering what fluids from the cisterna magnum would have shown in these cases. It seems logical to suppose that cistern fluid would be most likely to give a tumor picture in lesions of the posterior fossa. Unfortunately, we have no laboratory data at present on fluids obtained by cistern puncture, although Dr. Cushing has often remarked that the cisternal fluid encountered at operation in acoustic tumor cases is grossly xanthochromic when the ventricular fluid from the same case is clear.

In the list of proved cases there were thirteen instances of chronic arachnoiditis which were represented by five lumbar fluids and eight ventricular fluids. A negative spinal fluid, in the presence of symptoms simulating brain tumor, led to a diagnosis of chronic arachnoiditis, which was proved to be correct in all the cases in which the spinal fluid was examined, and in all but two from which ventricular fluid was obtained. We have found no variations in the chemical content of the cerebrospinal fluids from these cases. There appears, however, to be a tendency toward a slight increase in the white cell count; i. e., an average of 7 white blood cells per cubic millimeter (all mononuclears).

It is of importance to note that all the proved negative cases gave negative laboratory findings. Thus far the errors have all been in making negative serologic diagnoses when a tumor was present rather than making positive diagnoses when no tumor was found. This seems

to be of considerable practical importance, as one may feel sure there is a lesion present when the value of the various components of the cerebrospinal fluid is definitely above high normal.

Just what mechanism is responsible for the changes observed in the cerebrospinal fluids in cases of tumor is problematic. We believe that the greater part of the picture is probably due to an increase in protein caused by an exudation around the tumor; therefore, the larger the tumor mass and the more intimate contact it has with the cerebrospinal space, the more marked the chemical variations in the fluid will be.

SUMMARY

1. The normal ventricular fluid differs markedly from the normal spinal (subarachnoid) fluid in one respect only, namely, in its total protein content, which is from three to four times as great in the spinal fluid.

2. The picture presented by spinal fluid in brain tumor is marked by a low cell count, a high total protein value, a characteristic gold curve (Type III) and no significant variation in sugar content.

3. The changes in the ventricular fluid are in the same direction as in the spinal fluid. The significant points are therefore an elevation in total protein content above normal and a gold curve characteristic of that total protein value (Type I). A low cell count and a normal sugar content accompany the picture.

4. In chronic arachnoiditis both spinal and ventricular fluids consistently showed no deviations from the normal.

5. All fluids from cases in which no lesion was found gave negative laboratory findings. Positive results therefore indicate the presence of some pathologic lesion, although negative results, as in the case of chronic arachnoiditis, do not exclude a pathologic process.

6. A larger incidence of correct diagnoses occurred in spinal fluids (75 per cent.) than in ventricular fluids (42 per cent.).

7. The analysis of fluids grossly contaminated with blood is of no value. Whenever the red cell count approximates 2,000 cells per cubic millimeter, the value of the analysis is questionable; if the total protein content and gold curve are such as not to be accounted for by the contaminating blood, it is safe to assume that some lesion is present.

Clinical and Occasional Notes

DIMINUTIVE VISUAL HALLUCINATIONS IN A HYSTERICAL CONVICT*

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This interesting subjective phenomenon is characterized by the appearance of figures, minute in size and usually living, but always with surrounding objects that are seen in their proper proportions. And while the observed tiny creatures may be picturesque, fantastic or grotesque, they are never terrifying, but are prone to induce a pleasant or a humorous frame of mind. The phenomenon should not be mistaken for micropsia, a combined subjective and objective manifestation well known in ophthalmology, and which is an illusion in which real objects are seen, all of them uniformly reduced in size.

It is stated that the experience of diminutive visual hallucinations is first mentioned in the "Lives of Saints," in which Father Macaire, of the fourth century, told of having seen many little black creatures playing unseemly pranks on some priests. For years there have been occasional case reports showing this condition, and writers of fiction have had recourse to the phenomenon. Recently, Leroy of Paris has in several articles considered the subject under the title of "Lilliputian Hallucinations," a name suggested by Dean Swift's tiny folk in "Gulliver's Travels." While appreciating the effort of Leroy to keep before us a matter of consequence, the term Lilliputian seems fanciful rather than scientifically descriptive, so that the older designation of diminutive hallucinations appears preferable.

REPORT OF A CASE

The man who is the special feature of this paper spent some time in prison and a short period in a hospital for mental diseases. His father, a Spaniard, was said to have been a "bad man" and had died in the Norristown State Hospital. The mother was a mulatto. There were three brothers and two sisters. One brother was arrested twice for alleged infractions of the law, and a cousin died of some mental disease.

The patient, aged 33, had been a cook, was married and had one child. He was frail in build, superstitious, highly emotional and stammered a great deal.

Criminal Record.—At 26 years of age, the prisoner was sentenced to the Eastern Penitentiary for stealing a horse. He had previously been arrested on a similar charge, but had not been convicted. After two and a half years' detention in the prison, he was placed on parole, but this was subsequently violated. He was returned on the charge of incestuous rape. Two years later, the prisoner was again placed on parole, and, failing to keep the parole department informed as to his whereabouts, he was for a time lost from sight, but was finally found to be working in Camden.

Clinical Record.—About three years after entering the penitentiary, the prisoner became distinctly psychotic—briefly, he was hysteroid, paranoid, had

*Read before the Philadelphia Neurological Society, Oct. 24, 1924.

convulsive seizures and was generally unmanageable. The diagnosis of prison psychosis was made. He was removed to the Norristown State Hospital, where he responded to the change of environment, and in three months was restored and returned to the prison. Later, after, violating parole and being forcibly returned, he became very troublesome. He was believed to be an epileptic, and so was referred to me. The prisoner was having convulsive seizures at rather regular intervals, and because of the noise he made and the sympathy that was accorded him, he was given chloroform. To this he took kindly, but the necessity for its frequent administration made the matter burdensome. I had the opportunity of seeing the prisoner in one of his performances, which, from the convulsive aspect, was typically hysterical.

On entering the cell, I found the prisoner had already been placed in restraint by his cellmates. He was breathing forcibly but regularly, and was making a harsh barking sound. Following a convulsion, he would suddenly cease breathing, and would then tug violently at his restraint; after slight chloroform narcosis, the convulsions and the other motor disorder ceased; he laughed and became profane and obscene. This sequence of events was at times repeated once or twice. The prisoner had a remarkable psycholeptic manifestation as a part of the attack. He stated that always, being aware of the oncoming seizure, he begged his cellmates to place him in restraint. Then he felt cold and shivered, but recalled nothing further until the return of consciousness following the administration of chloroform. The prisoner said he fell in a trance, in which he found himself in a large room; the floor was covered with straw, and in the room were hosts of little men with exceedingly long fingernails, all wearing very large hats. The men carried chains that they rattled as they came running and shouting at him. There were also many tiny dogs in the room, and these barked as the little men sicked them on the prisoner. At the entrance door of the room stood the prison physician, who was his natural size, as were all the other surroundings; the prisoner called to the physician for help, but he only stood still and laughed. At last, he emerged from the trance, found himself in restraint, and when this was removed, he became lively, playful, profane and obscene. The little men and the little dogs never quite caught him. This psycholeptic manifestation had occurred many times; the scene was unvarying, and instead of causing terror, was regarded as humorous. The prisoner said that he experienced the scene whether or not chloroform was administered. The anesthetic was discontinued, and ultimately the hysteria yielded to treatment by judicious. systematic neglect.

Critical Review

INTRAVERTEBRAL TREAMENT OF DISEASES OF THE CENTRAL NERVOUS SYSTEM EXCLUSIVE OF CEREBROSPINAL SYPHILIS *

AN EXPERIMENT IN THE DISTRIBUTION OF DYES INTRODUCED SUBDURALLY

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Since the first intraspinal injection by a direct puncture was reported by Corning [1] in 1885, many diseases have been treated by injections of equally numerous medicaments. Intravertrebral treatment of but two diseases has proved to be of positive value, namely, meningococcic meningitis and tetanus. This communication does not contain details of the treatment of any disease. A brief outline will be given of diseases treated by the subdural administration of specific or immune serums of drugs, by drainage and irrigation and by miscellaneous procedures.

MENINGOCOCCIC MENINGITIS

In May, 1905, Jochmann [2] was working on a specific immune serum aginst meningococcic meningitis. In April, 1906, he reported, before the Kongress für Innere Medizin, the results of treatment in thirty-eight cases of epidemic meningitis. At first he employed subcutaneous and later intraspinal injections. About the same time, Kolle and Wassermann [3] reported that they had also prepared an immune serum. Flexner's [4] studies on an immune serum began a little after those of Jochmann in 1905. In 1906, he reported the discovery of a specific immune antimeningococcic serum which protected against experimental meningitis. In 1908, Flexner and Jobling [5] published a comprehensive report on serum treated cases.

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* Read before the Association for Research in Nervous and Mental Diseases, New York, Dec. 30, 1924.

1. Corning, J. L.: New York M. J. **43**:483, 1885.
2. Jochmann, G.: Deutsch. med. Wchnschr., 1906, No. 20, p. 788.
3. Kolle, W. E., and Wassermann, A.: Deutsch. med. Wchnschr., 1906, No. 32, p. 609.
4. Flexner, S.: Experimental Cerebrospinal Meningitis and Its Serum Treatment, J. A. M. A. **47**:560 (Aug. 25) 1906.
5. Flexner, S., and Jobling, J. W.: J. Exper. M. **10**:141 and 690, 1908.

Since then the specificity of this treatment has been accepted, and the mortality of meningococcic meningitis has been reduced from a figure varying between 42.5 and 90 per cent. to one of about 25 per cent.

The specificity of a serum depends on the particular strain of organism used in preparing it. Therefore, when Dopter [6] isolated an organism from the spinal fluid of a patient suffering with meningitis, that had all the morphologic and cultural characteristics of *Micrococcus intracellularis-meningitidis* of Weichselbaum, but which was not agglutinated by the usual meningococcus serum, it became necessary to produce serums of different valencies. Dopter called this organism the parameningococcus. Wollstein [7] corroborated Dopter's observations, and she found a number of intermediate strains. Gordon [8] found that the meningococci fell into four groups, which he designated as I, II, III and IV. I and III and II and IV were closely related. Since then a number of classifications have been made. At the Rockefeller Institute,[9] two main groups—the normal and paranormal—and two or more intermediate groups are recognized.

Since the recognition of the different strains of the meningococcus, all are agreed that a polyvalent serum should be employed pending the bacteriologic report. When the strain has been ascertained, opinions differ as to whether a polyvalent or monovalent serum should be used. It may be assumed that until it is proved that a monovalent serum has greater activity for a specific strain than a polyvalent serum against the same strain, a polyvalent serum has many advantages. All meningococcic serums sold in interstate traffic in the United States are now required to be polyvalent, with high titer against strains representing four different serologic groups. The successful treatment of meningococcic meningitis depends on the early recognition of the disease and its early treatment with a serum of high potency and specificity for the type of infecting organism.

During the early months of the war, the results of serum treatment were unsatisfactory, because the strains of meningococci causing the infection were, in the majority of cases, different from those employed in preparing the antimeningococcic serum used.

Efficient treatment is dependent likewise on proper dosage, frequency of injection, continuation of treatment and adequate distribution of the serum.

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6. Dopter, C.: Compt. rend. Soc. de biol. **67**:74, 1909.
7. Wollstein, Martha: J. Exper. M. **20**:201, 1914.
8. Gordon, M. H.: Great Britain National Health Ins. Med. Res. Com. Spec. Rep., Series **3**:10, 1917.
9. Blackfan, Kenneth D.: Medicine **1**:139, 1922.

Because this paper does not describe in detail the measures used in the treatment of meningococcic meningitis, it must not be understood that the intraspinal administration of serum constitutes all of the treatment. For example, intravenous injection of serum is strongly recommended by Herrick [10] and many others.

Whatever else may be done, the serum should always be introduced subdurally. Since it has been pointed out by Sophian [11] that symptoms of collapse are due to sudden increase of intracranial tension, the amount of serum injected should be slightly less than the amount of fluid withdrawn. The gravity method of injection described by Heiman [12] is the one of choice. The following doses are considered safe at different ages: One to five, 5 to 15 c.c.; five to ten, 10 to 20 c.c.; ten to twenty, 20 to 30 c.c., and over twenty, 30 c.c. or more. When, because the fluid is viscid, little may be removed by spinal puncture, small doses frequently repeated are necessary. The interval between injections should not exceed twenty-four hours. In severe cases, the injection may be repeated every eight to twelve hours for three or four doses. One objection to frequent injections is the production of a localized meningitis, with resulting long lasting pain and paralysis of adjacent nerve roots.

With the administration of the serum, the meningococci become reduced in number and altered in size and staining property. The extracellular organisms become engulfed and finally disappear. Their viability is reduced, and they can no longer be grown on culture mediums.

The treatment should be continued until the clinical signs disappear, with a change to normal clearness of the fluid and freedom from organisms. Usually the two go hand in hand. At times a turbid fluid may be present when all clinical signs have disappeared, and this may be the result of the injection of a foreign protein.

Other routes than the endolumbar are at times necessary for the introduction of the serum. The injection of serum directly into the ventricles was first employed by Cushing and Sladen.[13] Lewkowitz [14] especially advises intraventricular injection from the beginning of treatment, but except in complicated or severe cases it is not generally in use.

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10. Herrick, W. W.: Cerebrospinal Meningitis, J. A. M. A. **71**:612 (Aug. 24) 1918.

11. Sophian, A.: Epidemic Cerebro-Spinal Meningitis, 1913, St. Louis, C. V. Mosby Company.

12. Heiman, H., quoted by Sophian, Footnote 11.

13. Cushing, H., and Sladen, T.: J. Exper. M. **10**:548, 1908.

14. Lewkowitz, K.: Arch. de méd. d. enf. **12**:22, 617.

When a block in the cerebrospinal system occurs, because of a heavy exudate or meningeal adhesions, the intraventricular as well as other routes of injection must be employed. Because of the lack of danger and ease of cisterna magna puncture, as described by Wegeforth, Ayer and Essick [15] and amplified by Ayer,[16] injections in the dorsal and cervical regions are unnecessary.

Prior to the use of a specific serum, the mortality of meningococcic meningitis is given as from 20 to 75 per cent. by Hirsch,[17] who collected the statistics of forty-one epidemics. The figures compiled by Flexner show that the death rate in eighteen epidemics was between 42.5 and 90 per cent., varying in different epidemics and at different periods of the same epidemic. In treated cases, Jochmann in 1906 had a death rate of 27 per cent., Levy of 16.2 per cent. and 21.7 per cent. in two epidemics, and Flexner and Jobling of 25 per cent. in 393 cases.

TETANUS

Blumenthal and Jacob,[18] in 1898, first advocated the injection of antitoxin by means of lumbar puncture. During the past twenty-five years, many hundreds of patients have received at least some treatment by this route, and there has been a slow but steady growth of favorable opinion in regard to it. Roux and Borrel [19] in the same year suggested direct injection of antitoxin into the substance of brain or cord.

Ransom,[20] in 1901, stated that subdural injections acted practically the same as injections anywhere in the subarachnoid space. In either case, the antitoxin passed rapidly into the blood by way of lymph channels, and only a trace was left in twenty-four hours. Park,[21] after experimental work in guinea-pigs, stated that "the results with intraspinal injections were considerably better than with intravenous and those with intravenous injections did much better than those receiving subcutaneous injections. The units required by the intraspinal method were less than by the other methods. Repeated large injections did not give any better results than a single sufficiently large injection."

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15. Wegeforth, P.; Ayer, James B., and Essick, C. R.: Am. Jour. Med. Sc. **157**:789 (June) 1919.

16. Ayer, James B.: Arch. Neurol. & Psychiat. **4**:529, 1920.

17. Hirsch, Angus: Handbook of Geographic and Historical Pathology **3**:546, 1886.

18. Blumenthal and Jacob: Berl. klin. Wchnschr. **34**:1079, 1898.

19. Roux, E., and Borrel, A.: Ann. de l'Inst. Pasteur **12**:225, 1898.

20. Ransom, F.: Berl. klin. Wchnschr. **13**:336 (April 1) 1901; **14**:373 (April 8) 1901.

21. Park, W. H.: Tetanus, in Forchheimer: Therapeusis of Internal Diseases, Ed. 2, New York, Appleton & Co. **5**:466, 1915.

Adams and Horder [22] found "that antitoxin is readily and quickly absorbed from the theca into the circulation, so that the intrathecal route practically accomplishes in a short time all that can be attained by intravenous injection, with less risk of shock and with the possible, but unproven, advantage of some direct action upon the central nervous system."

Sherrington,[23] working on monkeys for the British Tetanus Committee, in 1917, reported that all the controls, all those receiving subcutaneous injections and all receiving intramuscular injections, had died, while 62.5 per cent. was the mortality for those treated intravenously and 27.7 per cent. for those treated intrathecally with tetanus antitoxin. Golla,[24] from experimental work on animals, found the intrachecal route indubitably superior, but later,[25] in a review of war hospital statistics, he said that he felt serum treatment in man had been a failure. He said, "By the time symptoms of tetanus appear, a sufficiency of toxin is already in the nervous system in such cases as would end fatally whether treated or not, whereas, in the milder cases, sufficient toxin to cause fatal tetanus is not manufactured." He concluded that if allowance is made for the modification of the disease due to prophylaxis, there had been no diminution of mortality.

It is extremely difficult to review profitably the results of the intrathecal treatment. Few patients have been treated by this route alone. Reported statistics usually do not differentiate the route of antitoxin administration. The well-known difference in virulence of the infection affords another difficulty.

Irons [26] urged the intraspinal injection of antitoxin in all cases of tetanus, and found the mortality in 225 patients treated with serum was 61.77 per cent., while that in twenty-one patients treated without serum was 85.7 per cent. The number of patients receiving intraspinal treatment is not tabulated. His figures showed that those receiving injections on the second and third days did better than those receiving them on the first day. This is due to the fact that the most acute and, of course, most unfavorable cases came to attention earlier. Park [27] argued that "there is no question that every hour counts and that those receiving intraspinal or intravenous injections within the first few hours of definite symptoms show a much greater percentage of recovery than those given in the table by Dr. Irons."

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22. Adams, F. W., and Horder, T. J.: Lancet **192**:682, 1917.
23. Sherrington, C. S.: Lancet **193**:964, 1917.
24. Golla, F.: Lancet **192**:686, 1917.
25. Golla, F.: Lancet **193**:966, 1917.
26. Irons, E. E.: The Treatment of Tetanus by Antitoxin, J. A. M. A. **62**:2025 (June 27), 1914.
27. Park, W. H.: Tetanus, Ibid., p. 469.

By far the largest series of cases studied is that of 1,458 reported from the British Army by Bruce.[28] These patients were treated in England, and therefore had comparatively long incubation periods. This final analysis is the summing up of several previous preliminary reports through which opinions on various points were evolving. The mortality was 34.8 per cent. Bruce emphasizes the difficulty in appraising the value of antitoxin once symptoms have begun. The fact that cases were treated in different ways, and that patients were suffering from other serious ills, such as wounds, fractures, septicemia, pneumonia, hemorrhage, etc., weakens the value of any tetanus mortality figures that may be obtained.

In spite of the teaching that if a lethal dose of toxin has been taken up by the nerves and is traveling toward the nervous centers before treatment is begun, no amount of antitoxin will save the patient, the British Tetanus Committee believe in giving the patient the benefit of the doubt and using large doses at the earliest possible moment by the intrathecal route. They recommend at least 24,000 units in twenty-four hours; 20 c.c. of high potency serum containing 16,000 units may easily be given intrathecally on the first and second day. This, if supplemented and continued by intramuscular and subcutaneous injections, should be sufficient.

They do not urge intravenous treatment because of the greater likelihood of producing anaphylactic shock.

ANTERIOR POLIOMYELITIS

The basis of serum treatment in anterior poliomyelitis rests on the observations of Römer and Joseph [29] that immune bodies are present in the blood of recovered cases. The experiments of Flexner and Lewis [30] showed that the intraspinous injection of an immune serum sometimes is effective in preventing experimental poliomyelitic infection in the monkey when intravenous injection does not. Flexner and Amoss [31] showed that similar protection was afforded when the virus was injected intraspinously and intravenously.

Netter [32] was the first to treat human cases, and he reported some success in the treatment of cases of the acute ascending variety.

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28. Bruce, Sir D.: J. Hyg. **19**:1, 1920-1921.

29. Römer, P. H., and Joseph, K.: München. med. Wchnschr. **57**:568, 1910.

30. Flexner, S., and Lewis, P. A.: Experimental Poliomyelitis in Monkeys. Seventh Note: Active Immunization and Passive Serum Protection, J. A. M. A. **54**:1780, 1910.

31. Flexner, S., and Amoss, Harold L.: J. Exper. M. **20**:249, 1914.

32. Netter, A.: Bull. de l'Acad. de méd. **71**:525, 1914.

Sporadic reports occurred until 1917, when Nuzum and Willy [33] reported 157 cases treated intravenously and intraspinously, injecting a total quantity of from 40 to 75 c.c. of a serum produced by immunization of animals with the streptococci cultivated from poliomyelitic cases. Amoss and Eberson [34] tested Nuzum's serum and found that it failed to show neutralizing or therapeutic power in the monkey when applied to their method against small doses of the virus of poliomyelitis. Under the same conditions, the serum of monkeys recovered from experimental poliomyelitis proved neutralizing and protective.

Although Rosenow used his immune horse serum intravenously, it is interesting to note that Amoss and Eberson were unable to find any evidence that it possessed effective therapeutic value in monkeys, or that it possessed the antibodies of the same nature as those present in the blood of monkeys which have recovered from experimental poliomyelitis.

Immune serums have been used by Amoss and Chesney,[35] Zingher,[36] and Le Boutillier,[37] who decided that immune serums were of some value. Peabody [38] is inclined to believe that no good case can be made out for serum in anterior poliomyelitis.

EPIDEMIC (LETHARGIC) ENCEPHALITIS

In 1918, Netter [39] recommended intraspinal injections of convalescent serums, with which he had seen good results in anterior poliomyelitis. He now feels that the long duration of the disease appears to render this treatment futile. The most serious attempt at treatment has been made by Rosenow, who grew a peculiar streptococcus isolated from the tonsils, teeth and nasopharynx of patients suffering from various forms of encephalitis. The serum of immunized rabbits and horses has been used by him in a number of cases, and favorable results in one-half of the cases is reported. The injections were made intramuscularly, intravenously and intraspinally. In a number of cases in which improvement was noted, the agglutinating titer of the paient's serum against encephalitis strains showed, twenty-four hours after the serum was given, a sharper increase than could be accounted for by the amount of serum injected. It is possible, therefore, according to Rosenow,

33. Nuzum, John W., and Willy, Ralph, S.: Specific Serotherapy of Poliomyelitis, J. A. M. A. **69**:1247 (Oct. 13) 1917.

34. Amoss, Harold L., and Eberson, Frederick: J. Exper. M. **28**:323, 1918.

35. Amoss, Harold L., and Chesney, A. M.: J. Exper. M. **23**:580, 1917.

36. Zingher, A.: Diagnosis and Serotherapy of Poliomyelitis, J. A. M. A. **68**:817 (March 17) 1917.

37. Le Boutillier, T.: Am. J. Med. Sc. **153**:188, 1917.

38. Peabody, F. W.: Boston M. & S. J. **185**:174, 1921.

39. Netter, A.: Presse méd. **28**:193, 1920.

that some of the good effects are nonspecific in character. The specificity of the organism has not been accepted, and the therapeutic results of Rosenow have not been repeated.

OTHER CONDITIONS

A few cases of recovered pneumococcic meningitis have been treated by pneumococcic serum by Cummings,[40] and Lamar.[41] Patients with influenzal meningitis rarely recover. Of thirteen patients who had recovered, one had been treated by convalescent serum intraspinally and one by autogenous and stock vaccine intraspinally. Neal[42] mentions a case of staphylococcic, one of influenzal and one of colon bacillus meningitis.

Bacigalupo[43] has reported two recoveries in three cases of tuberculous meningitis in which treatment consisted of intraspinal injections of tuberculin.

NONSPECIFIC SERUMS

As a result of their experiments regarding the antibactericidal properties of human serum, MacKenzie and Martin,[44] in 1908, injected from 15 to 20 c.c. of fresh human serum into the spinal canal of sixteen patients with meningococcic meningitis. Ten of these patients recovered. Since then a relatively large number of patients have been treated by autoserum injections. On the whole, the use of human serum has not been followed by brilliant results.

Pneumococcic meningitis has been treated by injections of antimeningococcic serum (Stainforth[45]); Hollis and Pardee[46] refer to the patient with tuberculous meningitis treated by Schaeffer in 1913 by the intraspinal injection of antimeningococcic serum, and report recovery of two patients with undoubted cases and two with doubtful cases following this treatment. Streptococcic meningitis has been treated by normal horse serum by McCarthy.[47] Following the report of Goodman,[48] Langley Porter[49] treated seven patients with chorea by intraspinal

40. Cummings, J. H.: Lancet **183**:1294, 1912.
41. Lamar, R. V.: J. Exper. M. **16**:581, 1912.
42. Neal, Josephine B.: Arch. Pediat. **38**:1, 1921.
43. Bacigalupo: München. med. Wchnschr. **62**:22, 1915.
44. MacKenzie, I., Martin, W. B. M.: J. Path. & Bacteriol. **12**:539, 1907-1908.
45. Stainforth: Arch. med. Belges **11**:248, 1912.
46. Hollis, A. N., and Pardee, J. H.: Recovery from Tuberculous Meningitis After Treatment with Intraspinal Injections of Antimeningococcic Serum, Arch. Int. Med. **26**:49 (July) 1920.
47. McCarthy, F. P.: Boston M. & S. J. **177**:621, 1917.
48. Goodman, A. L.: Arch. Pediat. **33**:649, 1916.
49. Porter, Langley: Intrathecal Injection of Horse Serum in the Treatment of Chorea, Am. J. Dis. Child. **16**:109 (Aug.) 1918.

injections of normal horse serum, with some improvement, but with no cures nor as striking results as those of Goodman.

Further experiences with autoserum are reported by Brown, Smith and Phillips,[50] who are favorably impressed with this method. Epidemic (lethargic) encephalitis was treated by Brill[51] by intraspinal injections of autoserum, and by Fendel[52] with an influenzal serum.

DRUGS

Sicard,[53] in his thesis, refers to the intraspinal injections of non-toxic solutions—sodium chlorid in dementia paralytica, potassium iodid in cerebrospinal syphilis and potassium bromid in epilepsy—all without result. In 1902, Seager[54] recommended compound solution of cresol in the treatment of meningococcic meningitis. Manges[55] tested its effect on a number of patients. Wolff[56] recommended protargin strong following the recovery of five of eight patients whom he treated. Coglievina[57] used Dispargin, a colloidal silver preparation, in a case of epidemic meningitis and in a case of streptococcic meningitis, both of which recovered. He quotes two cases of epidemic meningitis, one treated by Villard and one by Gaudeau, with a colloidal suspension of silver. Rocaz[58] used colloidal tin in a case of staphylococcic meningitis. Ethyuhydrocuprein hydrochlorid, a cinchona derivative, has been employed in experimental septic meningitis by Kolmer and Idzumi[59] and clinically in epidemic and septic meningitis by Friedman,[60] and Kolmer.[61] Isoctyl hydrocuprein dihydrochlorid, a similar cinchona derivative, has been employed in septic meningitis. This drug is reported to have a germicidal value four times as great as that of the former and no greater toxicity for the human body. Lewy[62] analyzed twelve cases treated by Linck, Zimmerman and Kurt Huenges. Six of the ten patients in the cases reported by Linck recovered, but in only one was there a positive culture, staphylococcus. Lewy reported seven cases. All of the patients who showed a positive culture from the spinal fluid eventually died. Only one of his patients recovered.

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50. Brown, J. A.; Smith, G. E., and Phillips, G.: Canad. M. A. J. **9**:52, 1919.
51. Brill, I. C.: Med. Rec. **97**:1079, 1920.
52. Fendel: München. med. Wchnschr. **67**:353, 1920.
53. Sicard, A.: Thèse de Paris, 1899-1900, No. 124, Carré et Naud.
54. Seager, H. W.: Lancet **163**:1188, 1902.
55. Manges, M.: Med. News **84**:913, 1904.
56. Wolff, G.: Deutsch. med. Wchnschr. **41**:1486, 1915.
57. Coglievina, B.: Wien. klin. Wchnschr. **29**:1148, 1916.
58. Rocaz: Gaz., hebd. d. sc. méd. de Bordeaux, **9**:218, 1920.
59. Kolmer, J. A., and Idzumi, G.: J. Infect. Dis. **26**:355, 1920.
60. Friedman, H.: Berl. klin. Wchnschr. **53**:423, 1916.
61. Kolmer, J. A.: Therap. Gaz. **44**:697, 1920.
62. Lewy, Alfred: Ann. Otol., Rhinol. & Laryngol. **33**:254, 1924.

Unfortunately, the use of drugs in the treatment of meningitis is without avail. Flexner and Amoss [63] tested the bactericidal power of a given strength of compound solution of cresol and protargin strong both in vivo and in vitro, and found them to be without effect and "the chemicals have shown themselves not to be curative but rather to be injurious."

In 1906, Meltzer and Auer [64] called attention to the possible value of magnesium sulphate as an adjunct in the treatment of tetanus. They recommended injections of 25 per cent. aqueous solution, giving 1 c.c. for each 10 kg. of body weight. Meltzer thought that death occurred as the result of severe and exhausting spasms which could be controlled by magnesium sulphate. This procedure is attended by real danger of paralyzing the respiratory center and stopping the heart. Robertson [65] has presented a careful study of the merits of this treatment. He collected eighty-one cases, in all of which the patients had received magnesium sulphate in addition to other treatment. The mortality was 44.4 per cent. He found the subcutaneous method of injection distinctly safer, and concluded that "there can be no doubt in the minds of those who review the evidence that in magnesium sulphate we possess a most valuable addition to our armamentarium in the treatment of tetanus." However, Bruce,[66] in his final report of the British series, says that the use of magnesium sulphate in the Home Hospitals proved a failure.

It may be mentioned that Leonard [67] reported favorably on the use of intraspinal injections of magnesium sulphate in twelve cases of delirium tremens.

A few isolated reports may be found on the treatment of other diseases, as epinephrin in anterior poliomyelitis by Lewis.[68]

IRRIGATIONS

Horsley [69] treated one moribund patient who had meningitis with spinal subarachnoid irrigations of a weak mercuric chlorid solution, without result. Since that time, a number of others have performed irrigations with physiologic sodium chlorid solution and solutions of antiseptics, usually without result. Formachidis [70] has recently reported

63. Flexner, S., and Amoss, Harold L.: J. Exper. Med. **23**:683, 1916.

64. Meltzer, S. J., and Auer, J.: J. Exper. Med. **8**:692, 1906.

65. Robertson, H. E.: The Present Status of Magnesium Sulphate in the Treatment of Tetanus, Arch. Int. Med. **17**:677 (May) 1916.

66. Bruce, Sir D.: J. Hyg. **19**:1, 1920-1921.

67. Leonard, E. A.: Intraspinal Injections of Magnesium Sulphate in Delirium Tremens, J. A. M. A. **67**:509 (Aug. 12) 1916.

68. Lewis, P. M.: Med. Rec. **90**:541, 1916.

69. Horsley, V.: Brit. M. J. **2**:1286, 1890.

70. Formachidis, quoted by Blackfan, K. D.: Footnote 9.

successful treatment in a case of epidemic meningitis by irrigations of the spinal cavity with physiologic sodium chlorid. Weed and Wegeforth [71] have shown that the chemical irritation even of this solution is so great that death followed from respiratory failure in a high percentage of experimental animals. In another series of animals with experimental meningitis, Wegeforth and Essick [72] irrigated the spinal canal with many of the commonly used antiseptics and found that none prolonged life.

SPINAL PUNCTURE

The majority of those who recovered from influenzal meningitis in the cases reported by Neal, and from pneumococcic meningitis as reported by Lamar, were treated by lumbar punctures alone. Royster [73] reported two additional cases of pneumococcic meningitis. In other cases of meningitis reported by Kuemmell, Netter, Tedesco,[74] and Bourges,[75] the treatment consisted of repeated lumbar punctures. Because of unsatisfactory serums in use in England during the early months of the war, some authorities stated that the old method of lumbar puncture was the better form of treatment for epidemic meningitis. Foster and Gaskell [76] especially recommended lumbar puncture. Olitsky,[77] in an epidemic in southern China in 1918, had the opportunity of seeing patients who received no treatment and those in whom lumbar punctures alone had been performed. The mortality rate without treatment in 104 cases was 84.6 per cent.; with repeated lumbar punctures in 346 cases, it was 54.1 per cent. In view of the unsatisfactory reports from others, one is inclined to believe that the diminution in deaths may have been due to other reasons.

Following the observation of Steinbach [78] that the course of delirium tremens was shorter and milder following lumbar puncture, the procedure has been employed in a number of cases. Hoppe [79] believes that lumbar puncture offers a valuable aid in the treatment of acute alcoholism and delirium tremens. Occasionally lumbar puncture has

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71. Weed, L., and Wegeforth, P.: J. Pharmacol. & Exper. Therap. **13**:317, 1919-1920.

72. Wegeforth, P., and Essick, C. R.: J. Pharmacol. & Exper. Therap. **13**: 335, 1919-1920.

73. Royster, L. T.: Virigina M. Semi-Month. **22**:4, 1917.

74. Kummell, Netter and Tedesco, quoted by Dandy, Walter E.: J. Surg., Gynec. & Obst. **39**:760, 1924.

75. Bourges, H.: Rev. gén. de chir. et d. thérap. **30**:68, 1916.

76. Foster, M., and Gaskell, J. F.: Cerebrospinal Fever, New York, G. P. Putnam's Sons, 1916.

77. Olitsky, quoted by Blackfan: Footnote 9.

78. Steinbach, Richard: Deutsch. med. Wchnschr. **41**:369, 1915.

79. Hoppe, Herman H.: J. Nerv. & Ment. Dis. **47**:93, 1918.

been used in the treatment in status epilepticus and in the convulsions of eclampsia and uremia (Wilson [80]).

Lumbar puncture is of undoubted, if but temporary, value in diabetes insipidus resulting from hypothalamic lesions. A case of Herrick's [81] of four years' duration was reported in 1912, in which polyuria and polydipsia disappeared after lumbar puncture, to return after three years. A similar result was obtained in the case of Graham.[82] Tucker [83] reported a case with disappearance of the polyuria, and quoted the case of Cammidge. Hall [84] quoted three cases of Maranonand Gutierriz, in which the patients were all beneficially affected.

Lumbar puncture as a means of relieving increased intracranial tension following injuries to the head, especially those without skull fracture, has been widely recognized and was practiced in the late war (Leriché [85]). Albert [86] concluded that it was the only efficient treatment for basal skull fracture and for concussion. Jackson [87] called attention to its efficacy in preventing the sequelae of head injuries ordinarily grouped under the term "traumatic neurosis."

CONTINUOUS DRAINAGE

Continuous drainage is more properly a surgical subject. Dandy recently reviewed the literature and called attention to a small number of cures of various types of purulent meningitis, and proposed a modification of the Haynes operation. He reported four cases of streptococcic and staphylococcic meningitis in which treatment by cisternal drainage was given, with three cures and one death.

MISCELLANEOUS PROCEDURES

Among miscellaneous procedures may be mentioned injections of air or oxygen into the cerebral ventricles and subarachnoid space. Sharp [88] reported three recoveries in twelve cases of tuberculous meningitis, the diagnosis not being verified by bacteriologic findings.

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80. Wilson, W. T.: Lumbar Puncture for the Relief of Convulsions in Puerperal Eclampsia, J. A. M. A. **67**:742 (Sept. 2) 1916.

81. Herrick, J. B.: Report of a Case of Diabetes Insipidus with Marked Reduction in the Amount of Urine Following Lumbar Puncture, Arch. Int. Med. **10**:1 (July) 1912.

82. Graham, Evarts: Spinal Puncture in Diabetes Insipidus, J. A. M. A. **69**:1498, 1917.

83. Tucker, John: Am. J. M. Sc. **163**:668, 1922.

84. Hall, G. W.: Am. J. M. Sc. **165**:551, 1923.

85. Leriché, R.: Lyon Chir. **12**:342, 1914.

86. Albert, F.: Lyon Chir. **15**:328, 1918.

87. Jackson, Harry: Circulation of Fluid, J. A. M. A. **79**:1394 (Oct. 21) 1922.

88. Sharp, Edward A.: Artificial Pneumorachis in Treatment of Acute Infections of Meninges, Arch. Neurol. & Psychiat. **6**:669 (Dec.) 1921.

Treatment by lumbar puncture and subcutaneous injection of the patient's spinal fluid has been employed in the treatment of epidemic encephalitis by Regett.[89] Unfortunately, his favorable results have not been generally repeated.

AN EXPERIMENT IN THE DISTRIBUTION OF DYES INTRODUCED SUBDURALLY

Admitting the possibility of subdural treatment of diseases of the central nervous system, certain requirements must be met for its successful consummation. It is necessary to have a drug, serum or other agent which has a specific action of a desired character, an innocuous action on nervous tissue, and a method for its introduction so that it may be brought into contact with all parts of the central nervous system in a required concentration and adequate amount.

That the latter requirements have not been attained may easily be seen from the long and often illogical controversies between those who favor intravenous and those who favor intraspinal methods of treatment of syphilis of the central nervous system.

Many investigations have been made of *intra vitam* staining of the central nervous system, in which it has been pointed out that certain dye stuffs are distributed only over parts of the central nervous system following their endolumbar introduction. It is unnecessary for our purpose to discuss the question of whether the nervous tissue is or is not actually intravitally stained by such methods.

Dandy,[90] in his recent contribution on meningitis, stated that if a cubic centimeter of India ink replaces an equal amount of cerebrospinal fluid in the cisterna magna, the ink granules will be freely distributed throughout the most distant branches of the subarachnoid space (over the cerebral hemispheres) in less than one hour.

If the cerebrospinal fluid and its contents did not disappear, by whatever mechanism—absorption, diffusion or osmosis—no further search would be necessary for a proper route for subdural treatment. Unfortunately, as pointed out by Halliburton [91] and others, diffusible substances introduced into the subarachnoid space pass rapidly into the veins. Especially rapid is the diffusion of liquid in the subcerebellar district. Dercum [92] alluded to this fact as of great importance in pointing out the apparent fallacy of treating syphilis of the central nervous system solely by intraspinal methods.

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89. Regett: Presse méd. **28**:798, 1920.

90. Dandy, Walter E.: J. Surg., Gynec. & Obst. **39**:760, 1924.

91. Halliburton, W. D.: Brain **39**:214, 1916.

92. Dercum, Francis X.: Functions of Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **3**:230 (March) 1920.

Whatever dyes various workers may have used, they are all agreed that the meninges covering only certain parts of the brain are stained heavily by endolumbar injection. Goldmann,[93] who was particularly concerned with the study of the impermeability of the central nervous system to dyes introduced intravenously, was able to stain only the base of the brain, the cerebellum, the olfactory lobes, the optic nerves, brain stem and spinal cord by endolumbar injection. Following him, MacCurdy,[94] although not admitting the actual intravitam staining of the brain, found about the same distribution of the dye. Woolsey [95] found, following sublethal doses introduced subdurally, that the meninges in some areas were not more stained than by intravenous injection. One of us (L. J. P.)[96] corroborated the former results of the gross distribution of dyes. It was found that unless injected with lethal force or in excessive quantities, solutions of dyes introduced by the endolumbar route did not stain the cerebral hemispheres or the meninges covering them to any degree. When an injection was made subdurally over a hemisphere, the stain reached the meninges covering it, the brain stem and cerebellum, but was faintly and irregularly distributed over the opposite hemisphere and the spinal cord.

We wish again to point out that for the purpose of treatment we are not concerned with the eventual destination of small quantities of dyes or drugs, but with the rapid circulation of solutions of sufficient concentration, and the avoidance of dilution and absorption which occur as time elapses.

The most important communication relating to this subject is that of Solomon, Thompson and Pfeiffer,[97] who experimented with injections of neutral phenolsulphonephthalein in the lumbar subarachnoid space, the cisterna magna and the cerebral lateral ventricles. They pointed out that fluid introduced into one of these regions could be drawn to another region by aspiration. Solomon stated that when a lesion was cerebral, the site of election for injection was the subarachnoid space of the brain, or the ventricles. The experiments suggest that the displacement of the dye in the subarachnoid spaces is the result of diffusion and not of a true spinal fluid circulation.

When a solution of dye or drug is introduced into the subdural space, it is analogous to the introduction of a certain quantity of fluid

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93. Goldmann: Arch. f. Klin. Chir. **101**:735, 1913.

94. MacCurdy, J. T.: Psychiat. Bull. **11**:1, 1917.

95. Woolsey, W. C.: J. Nerv. & Ment. Dis. **42**:487, 1915.

96. Pollock, Lewis J., and Cluney, W. T.: Am. J. M. Sc. **155**:857, 1918.

97. Solomon, H. C.; Thompson, L. J., and Pfeiffer, H. M.: Circulation of Phenolsulphonephthalein in the Cerebrospinal System, J. A. M. A. **79**:1014 (Sept. 23) 1922; Ann. de méd. **13**:57, 1923.

into a closed vessel practically full of liquid. If an Ehrlenmeyer flask be filled with water, corked and inverted, and if 5 c.c. of water be removed through a glass tube penetrating the cork and 5 c.c. of a solution of methylene blue introduced, the stain will remain in the neck of the flask for a long time, and complete diffusion will not have occurred even at the end of two hours. If, while the dye is being introduced through a glass tube, corresponding in a rough way to the spinal canal, water be permitted to flow out of the flask through an opening roughly corresponding to the position of the basal subarachnoid spaces, the dye will circulate freely into the lower part of the flask, then diffuse slowly throughout the remainder. If the rate of injection is greater than the outflow, only a small quantity can be introduced, and this will not diffuse much higher into the flask than in the first experiment. If a second glass tube be introduced through the cork, extending to the upper level of the fluid in the flask, and water be permitted to escape from it while the dye is introduced through the first glass tube corresponding to the spinal canal, the dye rapidly rises to the upper level of the fluid in the flask and quickly and thoroughly mixes with it. It may be seen that the diffusion of the dye is hastened by currents in the fluid into which it is introduced, and that the injection of the dye into a space almost filled with liquid does not result in rapid diffusion, because no active circulation of the fluid is produced. This we believe is, in a rough way, analogous to the conditions attending subdural injections into the cerebrospinal fluid. When injected by lumbar puncture alone, the drug or dye enters a space filled with an incompressible fluid and tissue compressible only to a slight degree. For this reason, circulatory currents are not produced by the injection.

We found, as before, that the injection of solutions of methylene blue by lumbar puncture alone failed to stain the cerebral hemispheres or their coverings except at the base. The cerebellum was freely stained, as was the brain stem. When the dye was introduced by cisterna magna puncture, the cerebellar fossae contained a greater amount of stain than before, and the brain stem was more deeply stained, as was the base of the brain, but the convexity of the hemispheres remained unstained. By injecting the dye subdurally over one of the hemispheres, we were able to stain freely that hemisphere and its coverings. The cerebellum showed a greater amount of stain immediately adjacent to the tentorium; the base was well stained, but the opposite cerebral hemisphere and its coverings showed very little stain.

Allowing the cerebrospinal fluid to escape from an opening in the dura over one hemisphere while the dye was being introduced by lumbar puncture, permitted the rapid distribution of the dye to *all parts of the central nervous system*. It may be said that the opposite cerebral

hemisphere was not as deeply stained, but the dye was found in large quantities. By this means a quantity of dye may be introduced without lethal effect such as would result from increased intracranial tension by intraspinal injection alone. It is well to refer to a former experiment in which it was found that when a subdural injection of a dye was made over a cerebral hemisphere and the fluid permitted to escape from a lumbar puncture, the opposite cerebral hemisphere was unstained, indicating that artificial circulation of cerebrospinal fluid is not produced as readily in this direction.

We propose that the only efficient method for reaching the hemispheres by subdural injection is to create an artificial circulation of the fluid by permitting it to escape through an opening in the dura over a hemisphere while an injection is being made into the lumbar subdural space or cisterna magna; that intraspinal, cisternal or cerebral subdural injections alone are inefficient.

News and Comment

FELLOWSHIPS IN NEUROPSYCHIATRY IN THE GRADUATE MEDICAL SCHOOL OF THE UNIVERSITY OF PENNSYLVANIA

The Commonwealth Fund of New York City has established five Fellowships in neuropsychiatry in the Graduate Medical School of the University of Pennsylvania. These Fellowships are for three years and are for the purpose of training candidates in conjunction with the Child's Guidance Clinic just established in Philadelphia. The course is similar to the three year course already established in the Graduate School, but during the last year special emphasis will be paid to child's guidance work. The stipend will be about $2,000 a year. Candidates must be between the ages of 25 and 35 years, inclusive, graduates of Class A medical school, and well recommended. Advanced standing will be allowed for those who show the proper credentials. Applications for these Fellowships should be made promptly to Dr. George H. Meeker, Dean of the Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa.

Abstracts from Current Literature

PRESENT TRENDS IN CLINICAL PSYCHIATRY. OSWALD BUMKE, München. med. Wchnschr. **50**:1595 (Nov.) 1924.

In this article, Bumke gives a comprehensive review of present day trends in psychiatry. As is true of so many of the older German writers, he uses such complicated phraseology that one not entirely familiar with his style may lose the more subtle meaning.

The early development of psychiatry showed marked Darwinian influences, which have continued to the present time. The future historian looking back on the mental trends of today no doubt would be irritated to confirm these Darwinistic, materialistic and mechanical conceptions, increasingly entwined with physical-romantic elements. The swing of the pendulum, especially since the war, has been from the entirely materialistic to the romantic. Metapsychology is at strife with materialism only for the purpose of ending its materialization, and the metapsychic so poorly harbors a pedagogic-rationalistic seed that one can even today see it being unmasked for want of a new explanation.

It cannot be denied that a combination of materialistic and real romantic presentations has been introduced into the psychologic and psychiatric literature of today. Most fantastic attempts at meanings and most heterogeneous observations are made and thrown together in literary colorings, and if one dare refute these doctrines, he is met with the explanation that the results are "found within." In face of this confusion, it is praiseworthy that there has been increased effort during the last two centuries to delimit the boundaries of psychiatric research and to study out anew its methodical rudiments.

It is certain that the double position of mental diseases, manifesting first psychic and then physical symptoms, and the certain urge bringing up the question of body and mind which is constantly facing psychiatrists, demands a vast amount of comprehensive clarification.

Bumke is most praiseworthy in his comments of Jaspers who has done so much to bring about a clear conception of psychiatry. Hönigswald states that psychiatry is a natural science and that even from a psychologic standpoint, it cannot be separated from the physiologic. This has recently been lost sight of, and serious attempts have been made to separate psychiatry from general medicine and to establish it as a branch of the mental sciences. This attempt has utterly failed from its outset.

What characterizes psychiatry most at present is its definite psychologic coloring. We no longer believe that the human psyche can be taken apart like a watch, and so experimental physiologic psychiatry has practically vanished from psychiatric clinics. However, it has been followed up by a psychology of coherence which is free from the ballast of brain mythology and the atomizing of the soul, endeavoring to know the mental complexes in the well and in the sick, and then not to pick them to pieces but to grasp them as a whole —a psychology that draws to the foreground in its observation, temperament and character, comprehension and reaction ability, tone and experience. It is psychology that governs psychiatry today, and here it does not merely register what it sees, but seeks out the oft-twisted roots of condition.

It is evident that an attempt to delve scientifically into the depths of the soul carries with it innumerable difficulties and dangers. Kraeppelin pointed out the sources of error that necessarily arise in searching the minds of the sick, and while Bumke does not deny these dangers, he emphasizes the fact that a psychopathology that would renounce such an investigation would perish. The mere fact that a sick person is not only reticent and moves slowly, but is also sad, contains in itself a subjective judgment, and between this and the exhibition of the sensitive illusion that all that happens affects him (Kretschmerz), there is a quantitative difference. We might admit that all sharply outlined observations in psychopathologic conditions arose in the same manner. They have neither to do with the brain mechanism of Wernicke nor with the apperception psychology of Wundt. What will remain will be the direct psychologic observations and our capability to establish these observations in an artistically alive form.

The author sees a danger again threatening us at this time, and that is of a psychology coming out of pathologic cases and entering into the healthy, confusing normal reactions with the diseased, and seeing the healthy person as a psychopath. If one follows the development of the schizoid, for example, during the last years, one must admit that by constant adulteration the pathologic aspect has entirely vanished.

It is Bumke's opinion that the danger of the modern psychologic trend of psychiatry is not so much the establishment of facts as the conclusions drawn from these facts. For years, as an observer of functional psychoses and psychopathic constitutions, he noted that these disease conditions always remain intelligible to the healthy, and then Jaspers emphasized the difference between these understandable or intelligible factors and the causative factors. But what holds true for the development of paranoia, for psychogenic reactions and hysterical constitutions, does not hold true for paralysis and schizophrenia. It was also a distinct advance when Bleuler recognized a definite form of thought disturbance in dementia praecox.

It is interpretations and not realities that we must combat in psychanalysis. This must be emphasized repeatedly. What the sick say and do and what the psychopathologists report of them no one has contradicted, but it is not necessary to believe some of the ridiculous interpretations of the psychanalyst. Schilder was of the opinion that facts and realities could not be refuted. The author admits this for facts and realities, but cannot subscribe to it for conclusions based on end-results. Bumke states positively that a science of facts as psychology and psychopathology cannot disregard the school of logic. Furthermore, he is convinced that psychanalysis has overstepped its bounds, and that in the not far distant future a quiet estimate of its historical value will be possible. But the success of the psychanalytic school was necessary in order that psychology and psychopathology would again concern themselves with their real problems. Further, this success was made possible only because the official science of psychology apparently knew so little of the actual spiritual workings, and handed stones instead of bread to those who actually wished to know more about the mind. The Freudian school contains so much that is repulsive that it would long since have perished had it not been for the fact that for a long time it was the only psychologic course which endeavored to take up the study of the mind as a whole. This is now past tense, and if the psychanalytic well is to run dry, we at least shall gratefully acknowledge that it has wiped away a lot of dusty requisites of a pure laboratory and writing desk psychology.

It is Bumke's contention that psychiatry will never be able to give up the psychologic position which it has reached today, and that the somatic problems deserve to be emphasized much more strongly than they have been in many places during the past years. Great progress has been made in serology and much has been said about internal secretions, the chemistry of psychoses, etc., but the neurologic or rather brain pathology has been sadly neglected. It deserves reflection when an investigator of the caliber of A. Picks, at the end of his academic activity and at the conclusion of extraordinarily great clinical service, deems it necessary to adjust and reestablish a methodologic study of neurologic research in psychiatry. Certainly the hopes that were attached to the name of Meynerts have not been fulfilled, and Wernicke's attempt to erect clinical psychiatry on the small foundation of aphasia study has been shattered for all time. But all this is not sufficient reason for us to abandon the means at hand for neurologic work.

It is noteworthy that the benefits which psychiatry derived from the study of numerous war injuries could have been very much greater had there been more psychiatrists interested in neurology. But the signs that the pendulum may now swing back are increasing. The psychic results of encephalitis in children have proved that everything which heretofore was accredited to the psychopath cannot be explained as hereditary-biologic, and that everything cannot be understood as psychologic. Or, expressed differently, the bounds of functional disturbance on the ground of new acknowledgments must be narrowed for the benefit of organic disease. This epidemic encephalitis permits us to speculate anew with the possibility that a localization of at least certain diseases may be successful.

Histopathology of the nervous system and anatomy of the psychoses do not play the same rôle as twenty years ago. Following the great results of Nissl and Alzheimer, histopathology led its own development into greater depths, and at the same time the psychologic course in psychiatry loosened a little the originally close connection of both disciples.

True the outlook on the possibilities of an anatomic foundation for psychiatry has changed. Twenty years ago, things looked simple in this regard. This was at the time the Kahlbaum observation method had reached its zenith. It was at the time when none less than Nissl described hysteria as a disturbance just as tenable as paralysis from a pathologic-anatomic point of view, and any attempt to indicate hysterical symptoms as psychologic he deemed unscientific.

Times rapidly changed. Soon Hoche described hysteria as purely functional in the sense that it did not and never would possess a pathologic anatomy. Hoche's doctrine was not well received and was looked upon as a phantom, not an ideal. The older of us well remember the ill favor and criticism of Hoche's endeavors of eighteen years ago, and yet the institution of Hoche's views was even then on the march.

In 1909, Bonhoeffer came forward with his exogenous syndromes. In the same year, the author, influenced by Hoche, endeavored to demonstrate that there were no disease entities for the functional psychoses; that all of these forms that were repeatedly pictured as inherited played into each other, and that their symptoms entwined and would remain combined; that they have always appeared psychologically understandable, but that they could not have a pathologic anatomy in the sense of a paralysis; that in the face of these facts it would be impossible to separate these diseases exactly, and that nothing would remain but to bring about the arrangement of types in systematic order. Bonhoeffer, as also the author, had already at that time advised the combining

of functional and exogenous syndromes, and, as one would say today, the talk of that time was of a multidimensional diagnosis.

In the meantime, it was seen more and more in practical work that the clinical school had reached its bounds of usefulness. If a single functional mental disease swallowed up all others, and if finally only the manic-depressive insanity remained of all other functional psychoses, it meant that the search for disease entities on the functional premises would have to be given up. But that conditions were just so for exogenous forms was evident from Bonhoeffer's further work.

How things changed is perhaps most clearly shown by a work of Kraeppelin's in 1920. Here not only Bonhoeffer's exogenous reaction forms are recognized, but thought passages developed that correspond in vast degree to those of Hoche's. It is also significantly shown that numerous expression forms of insanity are established through imaginary arrangements of the human organism and are exhibited all over in like manner where the preconception of the same are given.

In the meantime, clinical work brought forth good fruit, and the development of paranoia study came into its own. Here Specht, Friedman, Gaupp and others introduced a psychologic observation method that followed the genesis of insanity, and finally Bierbaum analyzed what had been set up as "Aufbau der Psychose" and structure analysis. We have long thought that certain psychopathic afflictions and certain psychoses might be due to endocrine disturbances (about which we know practically nothing). Kretschmer raised the question as to whether these endocrine disturbances did not serve as expression for certain body structures and whether there might not be a connection between body structures and character.

The biology of heredity carries the thought that behind the psychoses there are not disease entities, but a multitude of disease provoking causes.

Thus the question has completely changed in comparatively few years. Scarcely any of the dogmas of the clinical school remain. We know today that even paralysis may be cured and arrested. It has long been recognized that dementia praecox does not always lead to imbecility. But now the question arises whether the end results of schizophrenia are really anything more than a form of unfavorable departure that different mental diseases may take. That catatonic syndromes in the narrower sense are not specific, we have known for a long time; also that they are apt to follow any kind of injury, that qualitatively similar injuries would cause one brain to have a transitory psychosis and another to have a long lasting and even incurable defect, and that the picture in both cases would remain the same for a long time. It is conceivable that these things resemble the schizophrenic process. Here also the course would not depend on this or that disease entity, but, as in other pathology, on organism and disease producing causes.

The author has recently called attention to the similarity of certain schizophrenias to some symptomatic psychoses, not only so far as symptomatology is concerned, but with regard to the conditions of onset, and that there frequently is no difference between the two except that one results in recovery and the other in imbecility. This does not indicate that the two should be separated.

These questions will be answered only after zealous work is done in the fields of psychology, neurology, serology and anatomy, as well as in the fields of biology, inheritance and constitutional pathology.

The author is convinced that the distinction between the organic and functional must not be overdrawn.

MOERSCH, Rochester, Minn.

PATHOLOGIC CHANGES IN THE CENTRAL NERVOUS SYSTEM AFTER CARBON MONOXIDE POISONING. FREDERICK HILLER, Ztschr. f. d. ges. Neurol. Psychiat. **93**:593 (Nov.) 1924.

Investigations for the past hundred years have demonstrated a predilection of carbon monoxid for the lenticular nuclei. Practically all writers have included in the lenticular nucleus both putamen and pallidum, but the recent researches of Kappers, Spatz, Vogt and others prove the necessity of separating the pallidum from the putamen and caudate nucleus. Among the older contributors to the subject, the so-called lenticular softening in this disease has been chiefly pallidal softening. There have been two fallacies in past investigations, namely, the small number of cases and the concentration of attention on the pallidal pathology to the exclusion of study of other portions of the brain. Hiller's object is to show how important a study of the entire brain is in diseases which are supposed to have a predilection for certain definite portions of the brain.

The author reports the case of a woman, aged 27, with a psychopathic make-up, with a morphine habit of ten years' standing and a definite neuropathic heredity. She attempted to take her life by inhaling illuminating gas. Physical examination showed a cyanotic facies, dilated and regular pupils reacting well to light and convergence, normal reflexes and a negative blood and spinal fluid Wassermann reaction. During her stay in the hospital, the patient developed a bilateral ptosis and a convergent strabismus. After forty-eight hours, there was a bladder and rectal incontinence, and after five days, pneumonia developed. After nine days, a tremor of the extremities developed and on the tenth day, collapse and epileptiform convulsions of about thirty seconds' duration. There followed a paralysis of the left side, with conjugate deviation to the right and tremor of the face. On the eleventh day, all the extremities were paralyzed. Death occurred twelve days after the inhalation of the gas.

Necropsy showed petechial hemorrhages in the mucosa of the stomach, kidneys and bladder, severe pulmonary edema, beginning bronchopneumonia in the right lower lobe, bronchitis, an old aortic endocarditis and an old renal infarct. Gross examination of the brain and cord revealed extreme swelling of the vessels of the convexity with a pronounced arching of the right hemisphere. The sulci, especially in the parietal and frontal regions, were spread, and the convolutions smoothed out. Palpation revealed the probable presence of fluid. The meninges were greenish yellow. They were full at the base, and the vessels were normal. Over the central area was a meningeal hemorrhage. Section at this level showed numerous hemorrhages in the right hemisphere and in the cortex and medulla, with softening in between. In the basal ganglia, there was a sharply localized softening in the pallidum. The markings of the substantia nigra were not clearly defined.

Low power study with Herxheimer's fat and sheath stain and with Nissl's stain showed a marked dilatation of the pial vessels, which were filled with a homogeneous substance as in a beginning thrombosis. The pia was thickened about the vessels and deep in the sulci and was filled with blood in these regions. Areas of softening were found deep in the cortex and medulla. In the cornu ammonis, areas of softening were found in the deep portions and in the hilus of the fascia dentata. The caudate and putamen showed no changes. The pallidum, however, showed a definite area of softening which seemed to involve the entire pallidum, being bounded above by the internal capsule, ventrally by the anterior commissure, and laterally by the striatum. All three parts of the pallidum were affected, the lateral, mesial and internal

portions. In one part, the area of softening lay close to the anterior part of the substantia nigra. There was also an area of softening in the substantia nigra. The cerebellum was normal. The medulla oblongata and cord showed only the changes described above.

Detailed microscopic studies of the case were made.

Microscopic studies of the cortex showed changes of a diffuse and of a focal nature. The architectonics of the cortex showed no change. The ganglion cells were for the most part intact, but showed no more than the normal amount of fat. There was slight proliferation of the glia elements, with an increase in the fatty substance. The most striking feature of the diffuse cortical changes was a dilatation of the blood vessels, which were practically all filled with blood. Definite foci of softening were found almost always in the deepest cortical layers and at the boundary of the cortex and medulla. In exceptional cases, when the focus was very large, it extended upward to the fifth cortical layer. The foci were long and narrow, never round. Often these foci of softening coalesced. Histologic examination of these foci showed extreme uniformity. The cortical architecture in their vicinity was very much disturbed. In the foci and around it were usually numerous widely dilated vessels. Some of these were empty and some filled with blood or leukocytes, but none contained thrombi. The endothelial and adventitial elements were increased. In the adventitia were numerous gitter cells, many of which showed marked hyperchromatosis and pyknosis. Gitter cells were numerous elsewhere in the areas of softening, being most numerous in the center. Among the glia elements were many destroyed cells. Both glia and gitter cells contained much fat. A greenish-yellow pigment yielding no iron reaction was found in the foci. At the periphery of these foci, the glia elements became numerous, and among them the so-called Hortega cells were found in considerable numbers. The author uses this as evidence that both gitter cells and Hortega cells are sources of glia production. But while the foci of softening occur chiefly in the deepest layers of the cortex, the sixth, one can find evidences of beginning necrobiotic changes in the second layer of the cortex, with evidences of softening becoming marked only in the very deep cortical layers. In the second and third layers of the cortex, one may often find early foci which are marked by a lack of ganglion cells or by ganglion cells with vacuolated protoplasm and eccentric nuclei or even by cells with severe degenerative changes. The glia elements are about normal or somewhat increased. Many of the cells in the fifth cortical layer are similar to what Spielmeyer calls ischemic cell degeneration.

The foci of softening in the cornu ammonis had exactly the same characteristics as those in the cortex, and the areas affected are given above. The layer of giant cells was particularly affected and to a less degree than the polymorphous cells in the hilum of the fascia dentata. The large pyramidal cells were severely attacked and showed signs of degeneration and neuronophagia. There was intense glia proliferation in the molecular layer.

In the medulla itself were numerous hemorrhages, some punctate and some larger. Some of these involved the cortex and pia mater. Most of them were fresh. Numerous leukocytes were found around these hemorrhages while the ganglion cells in the vicinity showed degenerative changes, some of them severe.

Areas of softening were found in the basal ganglia. The most anterior focus of softening was found just at the beginning of the globus pallidus above the chiasm and did not cross the lamella separating the pallidum and putamen.

The putamen at this level was intact. Further sections showed two large areas of softening in the lateral portion of the pallidum and smaller areas in the mesial and internal portions. The putamen remained untouched. The microscopic picture of the foci was typical. Gitter cells, many of them filled with fat, were found in the center of these foci. New capillary formation and proliferation of the vascular endothelium and of the glia elements was also prominent. The vessels were dilated, but no thromboses or hemorrhages were apparent. The nerve tissue around the foci of softening showed a diminution in the large ganglion cells in many of which the Nissl substance was diminished. Dilatation of the vessels and moderate glia proliferation were present. No thromboses were found.

The claustrum, putamen, caudate nucleus, thalamus, internal capsule and the ventricular gray substance showed either no changes or most minute areas of softening.

A focus of softening was found in the red zone of the substantia nigra, with characteristics as described under the basal ganglia.

Several areas of softening were found in the cerebellum.

The midbrain showed no changes. The medulla oblongata showed small foci of softening in the nucleus of Burdach on one side and glia proliferation in the olives and in the substantia gelatinosa of the trigeminal nerve bilaterally. The spinal cord was free from any changes.

ALPERS, Philadelphia.

CISTERN PUNCTURES. JULIUS JANOSSY, Med. Klin. **21**:55, 1925.

Puncture of the cistern is not difficult, the method employed by the author being as follows: The head of the patient is bent forward slightly, thereby stretching the atlanto-occipitalis membrane. Then the occipital protuberance is palpated, as also the spinous process of the second cervical vertebra. At the edge of the under third of the distance between these two points, the skin is perforated exactly in the midline. The line which joins the point of puncture with the upper edge of the root of the nose points the direction of the needle. When one proceeds in this direction, the neck musculature is reached at the atlanto-occipitalis membrane, which is 4 cm. in thickness and a distinct hindrance. The membrane is cautiously perforated, whereupon fluid promptly appears if the patient is lying down. If he is sitting up, there is negative pressure in the cistern, and fluid appears only on suction.

The foregoing method is similar to that employed by Wegeforth, Ayer and Essick, but differs from that of Eskuchen. At first, the author used the Eskuchen method, as the American method seemed difficult. The American method is quicker, yet it requires more practice. Eskuchen's method is slower, but gives the operator a feeling of safety. To obtain the necessary experience and practice, it is important to try the puncture on cadavers, although it is much easier to perform on a living being.

Irreparable damage may be done by using incorrect methods. A lumbar puncture needle to which a syringe may be attached is best. It is advisable to use a needle with an accessory branch which may be closed.

Injury to the oblongata may be avoided by observing the following rules:

1. Puncture must be made exactly in the midline, and the needle must go in straight.

2. The head must be exactly in the middle position and remain so throughout the execution of the puncture.

3. The needle should not go in deeper than 6 cm. In the four cases of injury to the oblongata reported in the world literature, the needle went in more than 6 cm.

4. The patient should be in a lateral position. In the lying patient, fluid comes spontaneously on perforation of the atlanto-occipitalis membrane, and appearance of the fluid is warning not to go farther.

5. After perforation of the membrane, the needle should not go in more than 0.5 cm.

6. Injury to a cerebral vessel may cause life-long damage to the patient. Fortunately, there are not many vessels in this area. Several cases in which hemorrhage occurred are cited.

7. Fluid should be drawn slowly, especially when there is dislocation of the cistern due to tumor, abscess, etc., and if the tumor or abscess is not definitely localized, it is advisable not to draw off more than from 1 to 2 cm. of fluid.

A cistern puncture does not cause as much distress to the patient as does a lumbar puncture. Furthermore, it is not necessary for the patient to remain in bed following a cistern puncture; in fact, it is advisable for him to remain up.

The technic of the cistern puncture is easier than that of the lumbar puncture. The double puncture may be a valuable aid in the diagnosis of diseases of the spinal cord. If the lumbar canal is not obstructed by a tumor, the same kind of fluid is obtained from both punctures. In case there is an obstruction, the spinal fluid may differ from the cisternal fluid in its albumin content, refraction, color, etc. There may also be differences in pressure. If a neutral staining solution is injected into the cistern, it appears spotted in the lumbar sac or does not appear at all. For this purpose, various highly diluted neutral staining solutions may be used, such as methylin blue and phenolsulphonethalein. The author has used blood diluted with fluid, having first convinced himself that the spinal fluid did not contain any blood.

The author mentions Sicard's experiments with an iodized vegetable oil to determine obstruction in the spinal canal. Wartenberg has used other iodin preparations on dogs.

A case of spondylitis of the seventh dorsal vertebra is cited in which 0.75 c.c. of iothion was injected into the cistern following the puncture. The patient immediately grew very pale, was covered with cold perspiration, became dizzy, with rapid, weak pulse, and weak and slow respiration. Camphor and caffein were given subcutaneously, and in twenty minutes the patient began to improve. The roentgen ray showed an intensive shadow at the level of the seventh dorsal vertebra caused by accumulated drops of iothion. Incontinence and complete paraplegia followed, and only after ten days did the patient begin to improve, and he did not completely recover for six weeks.

The author states that theoretically one might expect important differences between cistern and spinal fluid findings in meningitis — that the fluid coming direct from the brain would be richer in leukocytes. Experiments have shown, however, that this is not true, and that in most cases there were more leukocytes in the spinal fluid.

Some authorities recommend cistern punctures for the relief of pressure, and undoubtedly patients endure under daily cistern punctures better than they do under lumbar punctures. The lumbar wash recommended by Podmaniczky in the treatment of meningitis could be made easier and more effective through the cistern.

Wegeforth, Ayer and Essick have employed the intracisternal serum method in acute meningitis, reporting one cure. Nonne reports one cure and Hartwich two by this method. In five patients, the author has used caffein diluted with 20 per cent. liquid solution, each time from 0.2 to 0.3 gm. Circulation remained the same; respiration became deeper and resembled the Kussmaul respiration.

An outstanding, interesting case was one of pernicious anemia that responded to the foregoing treatment. Lobelin obtained good results with the foregoing method in a case of acute morphine poisoning, and in this instance saved the patient's life.

The author has also used strychnin, epinephrin and atropin intracisternally, but it is as yet too early to have come to any definite conclusions regarding their value. Mention is merely made here that intracisternal medication may be employed in cases of poisoning, for example morphin, in beginning paralysis of the oblongata following lumbar anesthesia or narcosis and when there is a threatened paralysis of the respiratory centers.

In summary it may be said that the cistern puncture, performed with the necessary precautions, is no more dangerous than the lumbar puncture; the technic is easier, and it is also an improvement over the lumbar puncture from a diagnostic and therapeutic standpoint.

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COMPARATIVE ANATOMIC INVESTIGATIONS CONCERNING THE RELATIONS OF THE GLOBUS PALLIDUS TO THE SUBSTANCIA NIGRA. JOSEF WARKANG, Arb. d. neurol. Inst. an der Wiener Univ. **25**:195, 1924.

The globus pallidus differs from the putamen, the latter nucleus being related to the caudate nucleus. This opposition of the globus pallidus on the one side and the nucleus caudatus and putamen on the other, owes its origin in part to their different construction and in part to the different phylogenetic development of these two structures. Spatz has lately tried to go still further and show that the globus pallidus is not a part of the forebrain, but really belongs to the midbrain. His basis for this belief is based, in the first place, on the fact that in certain iron reactions iron can be demonstrated in the globus pallidus and in the substantia nigra, so that both these portions of the brain seem to have a similar chemical make-up. Again, the relation of these nuclei to the maintenance of muscle tone speaks for their similarity in function. The author believes, however, that these arguments are not valid, because the iron reaction is not specific for nerve cells, and the evidence for the maintenance of muscle tone by these nuclei is not well established. The most weighty argument in favor of the relation of the globus pallidus to the substantia nigra lies in the relation between the cell groups in these two regions. Mirto has observed certain cell relations between cells of the globus pallidus and those of the substantia nigra in the embryo. Spiegel, in comparative studies, showed that in many mammals groups of cells are to be seen going ventrocaudally from the globus pallidus. The question then arises whether these cells are in relation with the substantia nigra. The author attempts to answer this question by comparative studies in lower animals.

In man, a frontal section through the corpora mammillaria takes in both the globus pallidus and substantia nigra in one section. They are separated from one another by capsule fibers going to the pes pedunculi. These capsule fibers are not compact, but in Weigert preparations appear as interrupted, weakly staining, vertically running fibers. These fibers may mislead one

into thinking that they form a connection between the globus pallidus and the substantia nigra, since their staining reaction simulates that of the globus pallidus. These fibers are free from ganglion cells.

In *Cercopithecus,* a section at the level of the oral end of the substantia nigra shows the claustrum, putamen and globus pallidus well developed. The globus pallidus appears to consist of two portions which are bounded mesially by longitudinal fibers of the internal capsule. These fibers are arranged in bundles, and have between them a fiber-free substance which appears to be in connection with the globus pallidus.

Mesial to the capsule is the substantia nigra, and dorsally the corpus Luysii. Between both there appears a wedgelike mass which forms a continuation of the gray masses sprinkled in the capsule and stains similar to the globus pallidus. This mass, however, contains no large ganglion cells such as are characteristic of the globus pallidus and substantia nigra. Its small cells differ materially from the cells of the globus pallidus and substantia nigra.

In the orang-outang the arrangement is similar. In the lemur, both globus pallidus and substantia nigra are separated from one another so that both no longer appear together in frontal section. The caudal end of the globus pallidus is seen at the level of the tuber cinereum. The space which it formerly occupied is now taken by fibers running from the striatum and pallidum to the pes pedunculi. Between the fibers is a substance which continues on between the opticus and pes pedunculi and sends processes dorsally into the pes pedunculi. This substance is rich in cells which are not like the cells of the globus pallidus and substantia nigra. At the level of the corpus Luysii the globus pallidus has disappeared, and at a level of the oral end of the substantia nigra the light parts between the fibers of the peduncles have disappeared, so that a connection between globus pallidus and substantia nigra is impossible in this animal.

In *Hapale,* the relations are similar to those in the lemur. In *Pteropus,* a preparation at the level of the globus pallidus shows that the latter sends out a process consisting of large ganglion cells running between the optic tract and internal capsule to the tuber cinereum. There appears to be no connection, however, between the globus pallidus and substantia nigra.

In cats, in a section which cuts the caudal end of the globus pallidus, there is a gray body in the internal capsule which nowhere seems to be in union with the pallidum, but which has large ganglion cells similar to those of the pallidum. These cells seem to differ in no way from those of the pallidum. In a caudal section, this structure—the so-called nucleus of Meynert's commissure—seems to be a part of the globus pallidus. The most caudal portion of the globus pallidus is soon reached, however, before the corpus Luysii and substantia nigra are reached. Between the optic tract and pes pedunculi, one sees a lightly staining portion which, since it lies near the globus pallidus, would seem to be its continuation. These fibers, however, contain no ganglion cells. In cats, therefore, there seems to be no connection between globus pallidus and substantia nigra.

In dogs, the relations are the same as in the cat.

In pigs, the conditions are similar to those in carnivora. Here, however, as in *Pteropus,* we have a connection between the nucleus of Meynert's commissure, globus pallidus and tuber cinereum. The globus pallidus has disappeared before the substantia nigra is reached.

In rats, the globus pallidus and substantia nigra are not sharply defined. One sees cells of the globus pallidus lodged in the internal capsule. One sees

also a ventromedian continuation of the globus pallidus to the tuber cinereum, where the cells join with those of the nucleus paraventricularis. The substantia nigra is also not sharply localized, and shows numerous cells between the fibers of the pes pedunculi. All this leads to the assumption that there exists a union of the globus pallidus and substantia nigra in rats through the fibers of the pes pedunculi, but a union cannot be shown.

In *Cavia* also there appears to be a connection between the globus pallidus and substantia nigra. The same is true of *Macropus.*

In *Phoca,* the globus pallidus ends before the substantia nigra is reached, and the same is true in *Dasypus* and *Bradypus.* In *Ecludua,* there likewise seems to be no connection between globus pallidus and substantia nigra.

In summarizing, the author states that in the majority of mammals, there appears to be no connection between pallidum and substantia nigra. In only two animals, *Cavia* and *Macropus,* was the possibility of a connection definite. In a few animals, *Pteropus, Talpa* and rats, there was shown to be a connection between the globus pallidus and the tuber cinereum. In man and the higher apes, no union appears to be present. As one goes lower down in the animal series, the globus pallidus and substantia nigra become even more removed.

ALPERS, Philadelphia.

THE TREATMENT OF TETANUS WITH SPECIAL REFERENCE TO THE USE OF MAGNESIUM SULPHATE. CARROLL SMITH and W. E. LEIGHTON, Am. J. M. Sc. **168**:852 (Dec.) 1924.

Miolaier discovered the tetanus bacillus, "bacillus of the earth," in 1884. It was isolated by Kitasato in 1889, and the tetanus antitoxin introduced by Behring and Kitasato in 1890. But as yet the treatment is symptomatic and prophylactic because nothing has been found which will overcome the strong combination between tetanus toxin and nerve tissue. The prophylactic value of antitoxin is established, although Denati has shown that tetanus may develop after prophylactic serum as follows: (1) mild tetanus after a long incubation period, (2) local or partial tetanus and (3) fulminating tetanus, with a very short incubation period.

The reasons for failure are that a small number of persons are very susceptible; that there are at least three strains of toxic and one or more nontoxic strains of tetanus bacilli, and, finally, that the effect of the antiserum disappears after eight or ten days, and dormant bacilli or spores may become active after that period. The presence of these latent organisms usually follows the retention of a foreign body. Recurrence may be months after the first implantation, so repeated injections of prophylactic antiserum is urged; 1,500 units of serum repeated in ten days is the usual dose, but larger doses are advised by some, especially in the presence of a foreign body.

It has been recognized that antitoxin alone does not cure, that sedatives are necessary and that death is due to exhaustion, starvation or asphyxia during a spasm and not directly due to the toxin. All sedatives have failed to meet expectations. In 1899, Meltzer noted that magnesium salts given intracerebrally in rabbits was inhibitory. In 1905, Meltzer and Ant showed that the effect of magnesium salts was inhibitory and not stimulating in numerous animals. Intravenous, intramuscular and intraspinous magnesium salts were given with like effects. They next found that magnesium sulphate tended toward the control of the tetanic spasms induced in monkeys. A boy was cured of tetanus in 1906 by Meltzer, Ant and Blake by using intraspinal injections of magnesium

sulphate. Other authors have had similar results. The effect is purely symptomatic. The patients become quiet, the spasms become less or disappear, the spastic muscles relax, and the patients are able to take nourishment. The spastic muscles are affected before the normal ones.

In treatment, a 25 per cent. solution of chemically pure crystalline magnesium sulphate should be used subcutaneously, intramuscularly and intraspinously, but intravenously a 6 per cent. solution should be used. The solution can be sterilized by boiling or in the autoclave. Weston and Howard advise crystallizing the crude magnesium sulphate three times. With the subcutaneous method, 1.2 to 2 c.c. of a 25 per cent. solution to each 20 pounds (9 kg.) of body weight should be used four times in twenty-four hours. In severe cases, 16 c.c. should be used. The treatment should be repeated until all symptoms have disappeared. This is the slowest but least dangerous method. It may not suffice in severe spasms unless large doses are given. In intramuscular injections ether anesthetization should be used, then 2 c.c. of the 25 per cent. solution for each 20 pounds of body weight should be used. Effect may be obtained in less than one-half hour and last for two or three hours. The drug given in this way should be withheld for severe spasms. The intravenous method gives prompt effect, but this may disappear in half an hour. The myocardium is affected by too rapid injection, and respiratory paralysis may occur. This is an emergency procedure. A 6 per cent. solution given at the rate of 2 or 3 c.c. per minute and stopped with the beginning of relaxation or if serious complications arise is advised. The intraspinal method produces an effect in less than half an hour; the relief is longest, and lasts from twelve to thirty hours. Ether anesthesia is often necessary. One cubic centimeter of a 25 per cent. solution for each 20 pounds of body weight is used. The second dose should be smaller, about 0.8 c.c. to each 20 pounds of body weight. The doses mentioned are only suggestions and should be varied according to conditions. Relaxation should be partial and never complete.

The only danger seems to be respiratory paralysis, which may be relieved by giving slowly a 2.5 per cent. solution of calcium chlorid in physiologic sodium chlorid intravenously. From ten to fifteen cubic centimeters given intramuscularly and repeated if necessary may slowly neutralize the magnesium sulphate effect. Physostigmin in 1 mg. doses is sufficient to combat the effect, according to Joseph and Meltzer. Artificial respiration, especially the intratracheal insufflation of Meltzer and Auer, may be necessary. In the intraspinal method calcium chlorid is not sufficient, but the spinal canal should be washed out with Ringer's solution or with physiologic sodium chlorid. There may be urinary retention and hyaline casts in the urine, but these are temporary complications. After intravenous injection, there is considerable hyperglycemia with a mild glycosuria, both of which are temporary. Meltzer advised the following procedure:

1. Use the subcutaneous method every six hours as a routine.

2. In severe spasms, employ the intraspinal method and repeat if needed.

3. When there is an element of immediate danger, use the intravenous method.

4. When the intraspinal or intravenous method is not practical, give intramuscular magnesium sulphate with ether anesthesia.

A survey of the literature shows magnesium sulphate accepted as the best sedative to be used in tetanus. It should, however, never displace the use of the antitoxin as there is no evidence that the drug affects the tetanus toxin, while the antitoxin neutralizes the toxin not in combination with the nerve tissue.

The toxin may be given intravenously, intramuscularly and intraspinously. Stone recommends the following procedure: on the first day, 20,000 units intravenously (20,000 units intraspinously); on the second and third days, 20,000 units intravenously (20,000 units intraspinously); on the eighth or ninth day, 10,000 units subcutaneously or intramuscularly. Anaphylactic shock is rare, but if it does occur, ten minims of epinephrin chlorid usually relieves. Serum rashes occur in from 40 to 50 per cent.; usually they are relieved by 0.75 gm. of calcium lactate given intravenously.

The local treatment of wounds is important. They should be opened widely, and all secondary infection and necrotic tissue should be removed. Iodin used locally seems to have a specific action. Antitoxin should be given locally.

Eight recent consecutive cases of acute tetanus are then reported; all the patients recovered under treatment with magnesium sulphate and antitoxin serum. Each case illustrates the great value of magnesium sulphate. One case demonstrated also the greater efficacy of morphin when given after the use of magnesium sulphate.

WILSON, Clifton Springs.

THE SO-CALLED SPECIFIC EFFECT OF CONVULSION-PRODUCING TOXINS, PARTICULARLY OF TETANUS TOXIN, ON THE MOTOR GANGLION CELLS OF THE SPINAL CORD. ENRIQUE BARROS, Ztschr. f. d. ges. Neurol. u. Psychiat. **93**:720 (Nov.) 1924.

The changes produced in the motor ganglion cells by tetanus toxin has been a matter of discussion for a long time. Most authors agree with Goldscheider and Hatau that specific changes are produced in the ganglion cells, the intensity varying with the severity of the infection. Evidence for this belief is based on the fact that in animals to whom specific antitoxin has been administered, no such changes can be demonstrated. Other investigators, however, assert that there are no specific changes assignable to tetanus and that the changes in the motor cells are the result of normal involution processes or possibly of postmortem changes, particularly in cases which have been investigated some time after death.

There has been a good deal of work on the histologic changes in experimental tetanus. Beck described degeneration of the ganglion cells and peripheral changes in dogs infected with tetanus. Nissl described the same changes. Marinesco studied the cords of three porpoises, and concluded that the changes are dependent on the strength of the virus and the duration of the intoxication. He described degeneration of cells both in the anterior and posterior horns, characterized by pyknosis, dissolution of Nissl's substance and of the cell protoplasm. Goldscheider and Flatau studied the changes in the ganglion cells in 103 dogs and reported the following characteristic changes: destruction of the tigroid material, swelling of the nucleus, intense staining of the karyoplasm, executive nuclei, shrinking of the cell processes, clumping of cells and neuronophagia. Claude found changes similar to those described by Marinesco in a case of very slow intoxication. Vinzenzi described destruction or varicose atrophy of the cell processes of the cortical pyramidal cells as well as in the cells about the aqueduct of Sylvius and in the anterior horns. Daddi described rarefaction of the protoplasm in the cortical cells and swelling of the Purkinje cells in the cerebellum. Courmont, Joyou and Paviat studied three porpoises and three dogs. They concluded that the lesions produced are not constant and that they are independent of the occurrence of convulsions. Balus investigated the effect of various toxins on cell substance, among them being tetanus,

diphtheria and typhus fever. His conclusions were that tetanus toxin produced no specific effect but that the changes were due to a series of cellular, pericellular, vascular and neuroglial changes occurring in various infections. Some authors — De Buck and Demoor, Pechontre, Nageotte — have found the so-called specific changes in early cases of tetanus. Other authors have investigated the subject, but the changes have been lacking in uniformity. Just as in the foregoing cases the changes described have varied from slight or no lesions to definite lesions, such as severe injury of the tigroid material, cell processes and nuclei.

The changes found in human cases of tetanus vary a good deal. Goldscheider and Hatau describe two cases with the same changes as in animals: enormous and diffuse tigrolysis. Goebel describes similar changes. Westphal found only a swelling of the nucleolus in a few cells. Hunter described three cases, two with tigrolysis and one with no changes. Other authors have described tigrolysis in the cell of the anterior horns and a few have described changes in the cortical cells. On the whole, the evidence from postmortem study in man is almost similar to that in the experimental animal.

The author studied material from porpoises, mice and cats, all the material being properly controlled. Healthy animals were studied immediately after being killed in order to study normal nerve cells. Embryos were studied to prove that involution processes in nerve cells take place very early. Healthy animals were studied several hours after death in order to determine the effect of postmortem changes on the ganglion cells. Last of all, tetanus in animals was thoroughly investigated, some of the animals having a general infection, some local, and others being treated with antitoxin.

Sections were fixed in alcohol and liquor formaldehydi; various segments of cord were studied, and careful morphologic studies of all portions of the cells were made. The results of the investigation may be summarized for the sake of clarity, as follows:

1. There is a great difference between changes found in generalized tetanus infection and in tetanus intoxication. In a generalized infection, there may be no changes in the particular segment involved, even in the presence of convulsions. In an infection of long standing, however, definite changes are visible in the ganglion cells, but these are not limited to a local segment of the cord. In an intoxication, the ganglion cells of both anterior and posterior horns are affected. Definite and similar changes are found in the ganglion cells of the cortex.

2. The pathologic changes which cause the convulsions are not identical with those which are caused by the tetanus toxin, for these changes are found in areas which are not involved in convulsions and also in sensory areas. There is no evidence of a specific effect of the convulsion producing toxins on the anterior horn cells.

ALPERS, Philadelphia.

EXPERIENCES WITH ENCEPHALOGRAPHY. R. WARTENBERG, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:585, 1925.

Wartenberg believes that the indications for encephalography by means of air injection have been considerably enlarged during the past two years. He uses it in cases of post-traumatic neurologic and psychiatric disorders, chronic encephalitis, dementia paralytica, cerebral injury dating from early life and apoplexy, and even suggests its use in differentiating organic from functional hemiplegia. In his large experience he has had no fatal outcome attributable to the procedure.

He is accustomed to use the Bingel technic. Forty to 150 c.c. of air is introduced under slight pressure (50 cm. water) into the subdural space by lumbar puncture. The needle is fitted with a two-way stop-cock by means of which fluid can be withdrawn as the air is injected. The patient is kept in the sitting position during the operation to facilitate the rise of air to the cranial cavity. Care is taken to maintain the intraspinal pressure as near the original reading as possible, and as the air is introduced in increasing quantities the head must be kept very still to avoid headache and other disturbances. Except in cases of obstruction there seems to be no difficulty in outlining the ventricles of the brain. Great care is taken to have the head of the patient in exact position before the roentgen-ray exposure is made. No attempt is made to withdraw the air thus injected. Suboccipital punctures are performed in the lateral decubitus and seem to be accompanied by less subjective disturbance than the lumbar operation. If no air enters the ventricles from below, ventricular puncture is indicated. The most favorable position for making the exposure is the dorsal decubitus, the head lying on the plate.

Subjective complaints are frequent and occasionally severe. Headache is practically constant, lasting from two to four days, and greatly exaggerated by any brusque movement of the head. It is felt most in the frontal region or over the temple, and on the side of the brain which is diseased. Other patients noted that with change in position there was flowing of water within their heads, and this was readily audible to the examiner. Some patients vomited or broke out into sweats. Occasionally a seizure occurred in an epileptic patient soon after the injection. Slight rise in temperature and sleepiness were sometimes observed. Only twice did collapse supervene, and this was but temporary.

Subjective improvement occurred in a surprising number of cases, so much so that a few patients returned after an interval to have the procedure repeated. This was particularly noted in meningeal affections and occasionally in epilepsy.

The roentgen-ray findings are difficult to interpret in the present state of our knowledge. This is especially so in cases of tumor in which filling defects are present, but following destructive lesions of the brain there are also changes in the contour of the ventricles that are occasionally confusing. It appears that following trauma to the skull, even without fracture, there is often widening of the ventricle on the side of the injury, with rounding off of the upper angle of the "butterfly figure." This angle of the ventricle shows a tendency to "wander" toward the site of the injury, and in the illustrations this point is easily demonstrated. When the projection of the upper angle is toward the superior portion of the brain, signs point more to involvement of the superior portion of the cerebrum, and when it is bent on itself and extends toward the ear, signs point to involvement of the centers situated at an inferior level on the cortex. Following cerebral thrombosis or cerebral hemorrhage there are also alterations in the ventricles, chiefly widening of the affected side.

Serous meningitis often hinders filling of the ventricles, and shows a tendency in addition to divert the air into the subarachnoid spaces over the cerebrum. These spaces are often clearly shown.

Wartenberg has not performed encephalography in cases of brain tumor. The localization has been made by clinical means, and he believes that the risks are too great to run merely for the purpose of substantiating the diagnosis.

In the cerebral infantile palsies there is a constant widening of the ventricle on the affected side, together with large subarachnoid spaces which are readily visualized by means of the injected air. The pictures are almost characteristic in their confusion.

FREEMAN, Washington.

TRAUMATIC NEURASTHENIA. E. FARQUHAR BUZZARD, Mental Hygiene **8**:425 (April) 1924.

Buzzard's contribution illustrates how a strict organic neurologist may be driven to a psychiatric point of view by the inadequacy of his specialty in a difficult situation. The obstacle in this case is traumatic neurasthenia. Buzzard makes a determined effort to harmonize the interpretations of "organic" and "functional." Three main groups are described:

(1) *Anxiety Neurosis.*—These patients give a history of injury to some part of the body, the head perhaps being the commonest site. They differ in no respect from patients with anxiety neurosis seen in general practice without history of injury, or from the most common type of "shell-shock" victim met during the war. These patients complain of constant "pressure" headache, insomnia, fatigue, giddiness, tremors, inability to concentrate, loss of appetite, and pain and tenderness at the site of the injury wherever it may be. Under examination, their eyes blink continually, and voluntary movements are carried out feebly, hesitatingly and perhaps tremulously. The whole appearance is one of anxiety and misery, but careful examination reveals no evidence of organic injury.

(2) *Spinal Neurosis.*—This group comprises a much smaller number of patients, but the picture presented is characteristic. It may be labeled in descriptive language the "my poor back" group. The patient walks leaning on a stick in one hand, with the dorsum of the other placed over the lower part of his back. You know at once that the site of his injury is his spine.

Many of these patients have suffered only from a sharp attack of lumbago while making a physical effort. In addition to their characteristic attitude, they often present also the symptoms of anxiety neurosis. Examination reveals no sign of organic disease, but there is superficial tenderness over a great part of the spinal column.

(3) *Hysterical Neurosis.*—In this group the anxiety neurosis is often associated with some definite hysterical symptom, such as hemiplegia, monoplegia or paraplegia. These cases are not common, and their occurrence is immaterial to the question under investigation.

The general premises are that the development of neurasthenia has no constant relationship with the site or severity of the injury. It follows that direct injury to any part of the nervous system is not necessary for the production of neurasthenia. Buzzard feels that the determining factor is not to be found on the physical side, nor does hereditary nervous instability supply a satisfactory answer. The crux of the matter depends on whether or not the patient is responsible for the accident. If the patient is wholly responsible, then neurasthenia will *not* appear. If, on the other hand, responsibility may be shifted, then the soil is favorable for the seed of the traumatic neurosis. The seed may be supplied from various sources. Often it comes from the physician, the wife or some relative of the patient. Much neurasthenia will be avoided by the judicious and early application of psychotherapy. The physical manifestations of neurasthenia, be they ever so striking, are due to a chronic fear state. The medicolegal significance is made clear by the

following quotation: "As medical men, we are not in any way bound to go further than this. The final decision as to whether a condition such as I have described is to be legally regarded as the result, direct or indirect, of injury must rest with judges who interpret the law of the land."

There are two types in which there is a highly important organic element. The first is severe head concussion with retrograde amnesia. As a sequel there is vertex pressure headache, fatigue, depression and anxiety. Rest is both a preventive and a curative measure. In the "contusion" group, recovery is complete except for severe, paroxysmal headache, exactly localized at the site of the head injury. Even if roentgenography does not reveal bone injury, the skull should be opened.

STRECKER, Philadelphia.

ATYPICAL SPINAL TUMORS. NORMAN SHARPE, Am. J. Med. Sc. **157**:542 (April) 1924.

"Spinal tumors directly or indirectly involving the cord so often exhibit a similarity of symptoms and signs that a tumor syndrome has been evolved." This syndrome is composed first of pain and paresthesia, followed by muscle paresis, varying stages of anesthesia, muscle paralysis and impairment even to loss of sphincter control. It is generally understood that all new growths about or in the cord give rise to this group of symptoms and signs, chiefly, perhaps, because the larger number of tumors are situated posteriorly to the cord.

This so-called "typical" spinal cord tumor syndrome does not hold in a large number of cases. The order of appearance may be reversed; some symptoms may be omitted, and still further signs and symptoms may not suggest tumor but closely simulate some organic cord disease, such as multiple sclerosis, syringomyelia and myelitis. Symptoms difficult to explain may arise in distant portions of the nervous system, and the picture of organic cord disease may exist not only at the beginning, but also well on into the course of the new growth. "In consultations on spinal lesions it is not seldom that one hears the remark: 'As there has been no pain of any kind, we can safely rule out cord tumor.' It is unfortunate that this view is so widespread. More than half of the tumors involving the cord that have come under the author's observation were 'atypical.'"

The histories of four patients are then given to illustrate how atypical the course of a tumor may be. In Case 1, a woman, aged 28, complained of tremors and weakness in the legs. Later she noticed tremor of the hands. At this time a diagnosis of multiple sclerosis was made. She had general spacticity, a bilateral Babinski sign, tremor of the hands and a lateral nystagmoid twitching of both eyes. The upper abdominal reflexes were present, and there was no pallor of the disks. When seen six months later, the nystagmus had disappeared, and the anesthesias were more or less typical of cord tumor. There was no sphincteric loss, no pain, and the roentgenogram was negative. Operation showed an extradural tumor in the thoracic region. In Case 2 a youth, aged 16, received a mild injury of the back in 1916. In the autumn of 1919, he began to have slight pains in the legs. In March, 1920, he suddenly developed a complete flaccid paralysis to the groin except for the toes. There was impairment of sphincter control and diminution of pain and thermal sense. The diagnosis of multiple neuritis and transverse myelitis was made. Laminectomy disclosed an endothelial angioma springing from the connective tissue of the canal.

The third patient's condition was diagnosed cervical Pott's disease, and he wore a leather helmet for some time. He was also treated for syphilis, although the blood and spinal Wassermann reactions were negative. Laminectomy disclosed a circumscribed meningitis serosa resulting from the local infection in the third cervical vertebra. This case emphasizes strongly the value of exploratory laminectomy. The diagnosis was by no means certain until operation. The fourth patient, a girl, aged 14 years, began to lose power in the left leg and later in the right leg. As the right leg became weaker, pain developed in both shoulders and in the left hand. The bladder was next involved. The legs became spastic, and there was atrophy of the muscles of the hand. A diagnosis of spinal gliosis or syringomyelia was made. Later, a narrow band of hyperesthesia was found, as well as signs of lower sensory loss. Exploratory operations showed a large intraspinal lipoma.

In conclusion, the author points out that any condition regarded as organic cord disease should be watched constantly for signs of tumor. The early diagnosis of a tumor is important, and it is much better to operate and find an unrelievable condition than to allow a tumor to do irreparable damage to the cord.

WILSON, Clifton Springs, N. Y.

EXPERIENCES IN ENCEPHALOGRAPHY. O. FOERSTER, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:512, 1925.

Foerster communicates his experiences in the performance of more than a hundred ventriculographies. He has used lumbar puncture wherever practicable, but has occasionally had recourse to suboccipital puncture as well as to direct ventricular puncture. This last was used particularly in cases of cerebral tumor, and with great caution when signs pointed to tumor of the posterior fossa. The sitting posture is most favorable, although if the patient experiences severe headache, weakness or is restless, a somewhat oblique recumbent position may be substituted. A few whiffs of ethyl chlorid were given to children. It was well to bend the head of the patient backward and forward to promote the filling of the ventricles from below with air. The air was introduced as the fluid was withdrawn in small quantities, to the extent of 100 to 150 c.c., seldom more. In cases of ventricular puncture, 40 to 50 c.c. sometimes gave very good results. In congenital hydrocephalus 300 c.c. or more might easily be introduced. Pains in the back, especially in the scapular region were frequent, and served in one case to localize an extramedullary growth. Headache, vomiting and sweating were occasionally observed, but collapse very seldom. There were no deaths directly attributable to the injection. The most useful picture was the anteroposterior view with the plate beneath the occiput of the patient. Great care had to be taken to keep the patient's head properly fixed. Oblique views were worthless.

The variety of cases in which the method was used extended over a wide range of nervous diseases: hydrocephalus, infantile palsy, epilepsy, tumor, trauma, pseudotumor, encephalitis, etc.

Besides the encephalographic findings the author made estimations of the patency of the pathway of the cerebrospinal fluid by means of sodium iodid injected into the ventricles and tested for in the spinal fluid and in the urine. He encountered a number of cases of hydrocephalus in which the aqueduct and foramina appeared to be normal, the defect arising in the absorption of the fluid. In treating these cases he implanted a vein of the scalp into the subarachnoid space with gratifying results. He comments on the uselessness of the Balkenstich in such cases.

In vascular disturbances there was always dilatation of the ventricle on the side of the lesion.

In epilepsy with convulsions beginning in a particular part, even though there were no focal symptoms in the intervals, there was dilatation of the ventricle on the opposite side of the brain. Excision of the "trigger" area of the cortex was followed by long remissions. These cases also showed signs of arachnitis on the affected side.

In cases of tumor there was often difficulty in filling one of the ventricles, and distortion and dilatation of the opposite ventricle were frequent. Ventricular puncture was frequently used in these cases, and always when the signs pointed to involvement of the structures in the posterior fossa. Hydrocephalus was constantly observed in these last named cases.

The subarachnoid spaces were often enlarged in cases of pseudotumor owing to meningitis of either infectious or traumatic origin. The ventricles also showed considerable dilatation.

In epidemic encephalitis the author found dilatation of the ventricles. He believes that in this disease there are many indications of a disturbance in the circulation of the cerebrospinal fluid, and that this may be the cause of some of the symptoms of the disease.

In traumatic lesions the ventricle no longer retains its normal configuration, but extends outward in the direction of the lesion.

In concluding the article, which covers seventy-two pages and is illustrated with sixty-four photographs, the author says: "Encephalography is a kind of anatomy in vivo, and gratitude is due to him who first invented it."

FREEMAN, Washington.

TELANGIECTASIS OF THE SPINAL CORD. G. R. LAFORA, Arch. de neurobiol. **4**:320, 1924.

A case of telangiectasis of the spinal cord is described, diagnosed by other specialists as hematomyelia and sclerosis of the cord. Operation was performed successfully.

An officer of cavalry in the Spanish Army had fallen backwards from a horse. Two weeks after the accident, he began to notice a loss of sensibility and slight difficulty in the movement of his feet, and at the same time some retention of urine and feces. Motor difficulty increased afterward, and was accompanied by cramps in both legs. A lumbar puncture with abundant withdrawal of cerebrospinal fluid was followed by intense rachialgia and paresthesia in the legs, which lasted ten days. After this, there was slight improvement with hyperesthesia of the soles, which was replaced by hypesthesia.

At the time of examination by the author (two years after the accident), the patient suffered from paraparesis with hypesthesia and a sensation of stretching in the lumbar region. He did not experience erections for a month and a half. He had constipation and marked retention of urine, and sometimes incontinence. There were tonic cramps in the legs and feet while he was in bed or after he had been sitting for some time. The analysis of the cerebrospinal fluid gave 8 cells per cubic millimeter, a slight increase in globulin (Nonne and Noguchi tests showed traces; the Pandy test was slightly positive); increase in albumin (0.8 per one thousand) and negative Wassermann and Sachs-Georgi tests.

Neurologic examination showed that the legs were somewhat atrophied, especially the right. He had paraplegia which prevented walking without the

aid of a cane. Cramps in the legs and fibrillar tremor, especially in the left thigh, were produced with effort. The pupillary and tendon reflexes of the arms were normal; the patellar and plantar reflexes, weak; the Achilles, Babinski and cremasteric reflexes, absent; no clonus.

Touch and pain sensibility were greatly diminished in the zone corresponding to the third and fourth sacral roots. Thermic sensibility was preserved in the hypesthesic zones. Periosteal sensibility was lost from the third lumbar vertebra to the tibia, but was present in the ribs. Trophic disturbances were shown in loss of hair on the legs. Roentgen-ray examination disclosed a lesion at the fourth lumbar vertebra.

Since a post-traumatic arachnoid cyst was suspected, several punctures were made at different levels with the hope of puncturing the cyst. This treatment was not successful. Finally a laminectomy was carried out, and an elogated arachnoid cyst, placed above the region of the cauda equina, was found. The cyst moved freely during the respiratory movements. Its wall showed a thick bundle of varicose veins. The cyst was opened and the varicose veins were removed after double ligature.

After several days, there was improvement in the movements of the legs, the sensory disorders having disappeared. After a month, he could walk with more strength and steadiness. A relapse followed, and massage and reeducation of the muscles were started on account of a new display of spastic symptoms. The formation of a second cyst is regarded as probable by the author.

NONIDEZ, New York.

CONSTITUTION AND DISPOSITION IN PSYCHIATRIC RELATIONS. ALBERT M. BARRETT, Am. J. Psychiat. **4**:425 (Oct.) 1924.

This articles does not lend itself well to summarization, and should be read entire to appreciate the author's well knit account of the development of the conception of this relationship by various writers. Psychotic reactions are pointed out as arising out of the interaction of individual make-up and experience. Individuals differ in their susceptibility and manner of reaction to various noxious agencies and experiences. The "symptoms and course of the disorder are determined by factors inherent in the individual as well as in the environment in which he lives."

Bauer has said that variability of individual disposition to disease "rests upon the individual and temporal differences of the body states." Certain qualities are laid down at the moment of fertilization, and on these depends the constitution of the individual. The conditioning factors depend on multiform intra-uterine and extra-uterine influences, and adaptations of the organism. Both together determine body states and give the disposition to disease. Just what is inherited by way of quality, growth, tendency and manner of physiologic function we have as yet no means of knowing. Birnbaum has related the concept of constitution to psychiatry by way of general psychic reactions, functional capacity for work and psychic resistance power. Inadequacy is clinically distinguishable in the asthenic by way of lessened capacity for work, in the labile constitution by an undue lessening of capacity for resistance, and in the dissociative or schizoid make-up by incapacity in coordination and self regulation. Different disease forms or conditions preexist in these various constitutions. Various general factors predispose to psychic disorders, such as sex, age, race, climatic conditions, etc. The continually shifting interaction of normal physiologic functions brings about conditions which when acted on by suitable exciting factors induce psychic disorder.

Bleuler has called attention to physical and psychic interplay by way of flight into disease in the case of persons having organ inferiorities. "The physical creates an inclination to or necessity for a certain process. The psychic determines the time of its occurrence and the type of reaction." Bonhoeffer and Kleist have drawn attention to the tendency to delirious reactions following even slight infections in some persons; Lermann to the fact that psychic predisposition sometimes conditions abnormal behavior following epidemic (lethargic) encephalitis. Psychoneuroses are largely determined by individual constitution and disposition. Experience alone is usually inadequate to account for the symptomatology. Freud has stated that in more than one-half of his cases of severe hysteria, obsessional neuroses, etc., syphilis of the father before marriage was proved, which heredity "asserts itself in an abnormally strong and many sided instinctive life and a consequent sexual precocity." The epileptic, cyclothymic and schizothymic constitutions are considered in passing, and reference is made to Kretschmer's monograph on physical make-up and character along with Bleuler's theory of the combination of schizoid and syntonic constitution.

READ, Chicago.

THE SYNDROME OF THE HYPOTHALAMIC CROSSROADS. G. GUILLAIN and T. ALAJOUANINE, Folia neuropath. Esthoniana **3**:228, 1924.

The hypothalamic region is an extremely complicated collection of important fasciculi coming from the cerebrum, the cerebellum, the spinal cord and the brain-stem, as well as from the nearby structures. This has led the authors to denominate it the hypothalamic crossroads. In addition, there are certain centers concerning whose functions we are still much in the dark. Lesions in this area are not particularly frequent, and when they occur, they produce serious disturbances in motor and sensory functions. Guillain and Alajouanine assemble a group of cases in which the clinical findings were followed by postmortem examination, and other cases in which no anatomic examination was made. According to them, the following signs suggest a lesion of the hypothalamic region:

1. A hemiplegia that is always very mild, with slight exaggeration of reflexes on the side opposite the lesion and usually without a Babinski sign.

2. Choreo-athetotic movements which are as a rule more marked than in the typical thalamic syndrome, in which they are often lacking. The hand assumes a peculiar position with hyperextension of the fingers. The movements are increased by walking and by emotional disturbance, as in the parkinsonian tremor. Synkineses are uniformly present.

3. Sensory disturbances. Thalamic over-reaction and spontaneous pains are absent. Disturbance of "deep" sensibility is often present, but there is seldom any total anesthesia.

4. The cerebellar manifestations on the homolateral side, affecting chiefly coordination and tonus but sparing the static mechanism, are asynergy, dysmetria, intention tremor, and adiadokokinesis. Closing the eyes does not increase the disturbances. Cerebellar hypotonia is often extreme. Equilibration is undisturbed.

5. Hemianopsia is constant.

A single lesion giving these findings must involve the sensory radiation to the thalamus, the posterior portion of the internal capsule, the upper end

of the superior cerebellar peduncle and the optic tract. In this location, it must also affect the corpus Luysi "whence, perhaps, the hemiathetosis."

The lesion is usually a softening in a part of the area supplied by the posterior cerebral artery.

FREEMAN, Washington.

LOCALIZATION IN THE GANGLION SEMILUNARE OF THE CAT. W. F. ALLEN, J. Comp. Neurol. **38**:1 (Dec.) 1924.

Examination by Nissl's method of serial sections of the semilunar ganglion of cats in which one of the three trigeminal branches has been cut shows that there are two separate and distinct ganglia. A large cephalic and median portion consists only of ophthalmic-maxillary cells, and a smaller lateral and caudal division is made up entirely of mandibular cells. There is no mixture of the cells of the two ganglia at the point of their overlapping. In the ophthalmic-maxillary ganglion, there can be distinguished: (*a*) a narrow strip of purely maxillary cells cephalically at the base of the maxillary nerve, (*b*) a small area of ophthalmic cells, cephalically and medially, at the base of the ophthalmic nerve, (*c*) a mixture of both sorts of nerve cells throughout the rest of the ganglion, the ophthalmic cells predominating near the median border and the maxillary elsewhere.

A large number of ophthalmic cells at the base of the ophthalmic nerve are small cells, belonging apparently to the small and unmedullated type of the descriptions of Cajal and Dogiel. There is no definite localization of the alveolar inferior nerve and the lingual nerve cells in the mandibular ganglion. Cells from each of these nerves, usually somewhat bunched, are scattered throughout the entire ganglion. There are a few isolated nerve cells in the ophthalmic nerve, maxillary nerve, mandibular nerve and in the sensory root in close proximity to the ganglion. A number of isolated and small groups of cells appear in the motor root and immediately outside. Some of these may be proprioceptive cells having their peripheral processes distributed to the mylohyoideus and digastric muscles. (This last supposition has been confirmed in a research by Dr. Allen as yet unpublished.)

The human semilunar ganglion seems from existing anatomic descriptions to present the same localization in general as in the cat. If this is true, it is probable that the mandibular division of the ganglion, in cases of trifacial neuralgia involving this nerve, could be destroyed without injuring the ophthalmic-maxillary portion or the nerve fibers related to it or the motor root. Some suggestions regarding the procedure in such an operation are given.

C. J. HERRICK, Chicago.

THE HISTOPATHOLOGY OF EPIDEMIC ENCEPHALITIS. ISTVAN SOMOGYI, Ztschr. f. d. ges. Neurol. u. Psychiat. **93**:783 (Nov.) 1924.

Besides the usual findings which have been carefully described in encephalitis, the author has a new finding to report in a woman, aged 40, who, following a cold, developed pain in the right arm, the neck and then in the left arm. These pains lasted for two weeks, during which the patient was unable to sleep except after the administration of sedatives. On the fourth day of her illness, she became delirious. Her restlessness and sleeplessness were followed by lethargy. This, together with the delirium, disappeared eventually, and she returned home. Six months later, however, she returned to the hospital with a typical parkinsonian picture and with rapidly progressing bulbar symptoms.

She died after eleven months. At necropsy, slight convolutional atrophy, distention of the blood vessels, punctate hemorrhages in the lenticular nucleus, caudate nucleus and red nucleus were found. The left lenticular nucleus was smaller than the right. The hypophysis was definitely enlarged.

Microscopic examination of the brain sections revealed findings similar to those found in encephalitic or infectious diseases — broad and homogeneous media, overgrowth of the endothelium and new-formed capillaries. The capillaries were filled with blood, and many were thrombosed. Everywhere, but especially in the basal ganglia, there was round cell infiltration, and often plasma cell infiltration. The nerve cells of the cortex showed degenerative changes — chromatolysis, vacuolization, neuronophagia. These changes affected chiefly the cortical cells, but also the Purkinje cells of the cerebellum and their processes. There was also neuroglial proliferation.

All the changes described above are the usual findings in cases of encephalitis. In the author's case, in addition to these changes there were found round, light red, structureless bodies of varying sizes — from the size of a glia nucleus in the internal capsule to ten times this size in the basal ganglia. These bodies lay near or around the blood vessels, but most commonly in the lymph spaces. These bodies were of a homogeneous structure, and morphologically resembled most closely the chorea bodies described by Hudovering. They were of a colloidal nature.

ALPERS, Philadelphia.

OBSERVATIONS ON THE INFECTIOUS TREATMENT OF GENERAL PARALYSIS. M. NONNE, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:507, 1925.

In 1922 on an extensive tour of South America, Nonne was astonished to find that the infectious therapy of general paralysis was practically unknown. He notes with pleasure that the treatment is being adopted in many countries. He has treated 240 patients in his clinic, and of these eighty have been followed for two years or more. He has obtained complete remission of the disease in nineteen cases (23.7 per cent.). This means that the patient has returned to his occupation or profession apparently in full control of his powers. Nonne cites several instances of men of large affairs who have been able to take them up again. In addition, thirty-eight cases (47.5 per cent.) have shown arrest of the disease; twenty-three cases have not been benefited. There were seven deaths among the 240 cases directly attributable to the treatment. Nonne states: "Such frequent, long lasting remissions I never saw previously." The spontaneous remissions vary from 4 to 16 per cent.

Among the somatic symptoms improvement has been noted especially in speech defects, and in pupillary reactions. The cerebrospinal fluid varied in its reaction. Albumin and globulin were frequently reduced, sometimes completely suppressed. The pleocytosis was frequently reduced. In many other cases, the findings remained unchanged. The positive Wassermann reaction often persisted.

FREEMAN, Washington.

ENCEPHALOMEGALIA AND CEREBRAL TUMOR. P. ESCUDER NUÑEZ, Arch. de neurobiol. **4**:151, 1924.

This is a discussion of the differential diagnosis of encephalomegalia and cerebral tumor. The author emphasizes the fact that the encephalomegalia produced by the growth of a tumor is usually secondary to other symptoms which are manifested earlier and which depend on the involvement of areas of the

brain by the growing neoplasm. When these symptoms are carefully observed localization will be possible in a large number of cases. Cephalalgia, nausea and a general condition of hypertension appear later, and are the result of the increase of pressure brought about by the growth of the tumor.

In primary encephalomegalia, the symptoms mentioned above usually appear first, and those due to pressure on areas of the brain are manifested later or are not prominent. When a tumor grows in the vicinity of the ventricles or in the orifices of communication of the ventricles, the clinical picture of encephalomegalia may appear simultaneously with the symptoms due to its localization. With regard to the hypertension of the cerebrospinal fluid, the author states that it cannot always be interpreted as an index of increase in the amount of fluid, since compression by a tumor may produce a similar effect. Owing to this fact, he believes that no more than 5 c.c. of fluid should be removed when a tumor is suspected, even if the fluid flows in abundance, as, there being no increase in amount, it might lead to almost complete drainage of the ventricles. The law of growth followed by a tumor is also discussed.

NONIDEZ, New York.

THE QUESTION OF CEREBRAL OBESITY. INASABURO NAITO, Arb. a. d. Neurol. Inst. a. d. Wiener Univ. **25**:183, 1924.

This is a careful neurohistologic case report with mostly negative findings. A man, aged 40, a heavy drinker and smoker, had become progressively fatter since he was 19, weighing 181 kg. shortly before he died from pneumonia. He had had concussion of the brain at 3 and syphilis at 36, the Wassermann test remaining positive until death. The minute examination of the brain showed some dilatation of and hemorrhage in the third ventricle, with ependymal granulation and congestion, dilatation and perivascular hemorrhages in the ventricle wall and choroid plexus. There was slight increase of glia, no evidence of syphilis, and the nerve cells and fibers were normal. The same was true of the median and lateral nuclei of the thalamus (Malone) and the ganglia habenulae and mammilare, the center median, the nervus arcuatus, neuralis, reticularis hypothalami, reuniens and paramedianus. Chronic degenerative changes in Lenhossek's tuber ganglion and a gliosis and epithelial cell increase in the infundibulum were striking. Most striking, however, is the fact that the author makes no mention of the pituitary itself.

KRUMBHAAR, Philadelphia.

THE TREATMENT OF CEREBRAL SPASTIC PARALYSIS WITH SPECIAL REFERENCE TO THE STOFFEL OPERATION. FRANK D. DICKSON, J. A. M. A. **83**:1236 (Oct. 18) 1924.

Treatment of this condition is divided into three phases: (1) immediately after birth, consisting of repeated spinal punctures; (2) during the first three or four years following birth, when the treatment is entirely a matter of assisting the child to do what it can for itself, and (3) treatment of what might be called the chronic or final stage. In this last stage, the muscle training must be accompanied by such operative measures as are necessary to restore the capacity for action to the involved muscle groups. These operative measures are (1) tenotomy, (2) tendon transplantation, and (3) the Stoffel operation.

As a result of the author's experience with the Stoffel operation, he is convinced that it does reduce spasticity, relieve contractures and bring about local improvement.

NIXON, San Francisco.

EXPERIMENTAL INVESTIGATIONS OF THE RESORPTIVE FUNCTION OF THE CHOROID PLEXUS. R. PETERHOFF, Folia neuropath. Esthoniana **3**:110, 1924.

The author injected various substances into the lateral ventricles in a series of animals, and examined the choroid plexus to determine whether there had been absorption of the materials. The substances used were: grape sugar, hemoglobin solution, laked blood, pyrrol blue, carmin suspension, lithium carmin solution, trypan blue solution, potassium ferrocyanid, ferric citrate, and others. The time allowed for absorption varied within rather wide limits.

Microscopic examination of the brains showed that in no experiment was the injected solution taken up by the choroid plexus or by the ependyma. The meninges had taken up all the materials. The cortex had taken up the injected materials in only a few cases. When fixation was delayed for an hour after death, the Prussian blue reaction was positive in the epithelium and stroma of the plexus, but negative in the ependyma.

FREEMAN, Washington.

THE PROGNOSIS OF THE SEQUELAE OF EPIDEMIC ENCEPHALITIS IN CHILDREN. ROGER L. J. KENNEDY, Am. J. Dis. Child. **28**:158 (Aug.) 1924.

This study is based on sixty-one cases of residual encephalitis in children under 14 years of age. In many instances, the sequelae were observed for more than five years. The prognosis in the group that presented parkinsonism appeared to be particularly discouraging; of twenty-one patients, none improved, and slightly more than half became worse. When the principal symptoms are changes in personality and behavior, the outlook seems to be more hopeful; of twenty-three patients in this group, six improved. When the principal difficulty was a disturbance of sleep, it was found in nineteen cases that nine ultimately resumed normal sleep. The author includes also a group which presented as their chief complaint some respiratory difficulty; of seventeen children who had this sequel, two recovered, three improved and twelve remained unchanged.

VONDERAHE, Cincinnati.

INTRAMUSCULAR NERVE FINDINGS OF SENSORY TYPE IN THE SMALL INTESTINE, WITH A CONSIDERATION OF THEIR PROBABLE FUNCTION. F. W. CARPENTER, J. Comp. Neurol. **37**:439 (Oct.) 1924.

Intramuscular nerve endings which differ from the well-known motor endings and resemble others known to be sensory occur in the wall of the dog's small intestine as arborizations of exceedingly delicate varicose fibers compactly arranged. With the exception of a few in the subserous connective tissue, they have been found only in the external or longitudinal muscular layer embedded among the smooth muscle cells. The endings are noncapsulate. The slender unmyelinated varicose nerve fibers with which they are connected have been traced into the myenteric plexus. They may be the receptive terminals for (1) tonic and regulator reflexes of the musculature, (2) the myenteric or peristaltic reflex, or (3) they may initiate through adequate stimulation nervous impulses giving rise to sensations of pain. The last view is favored.

C. J. HERRICK, Chicago.

THE MALARIAL TREATMENT OF GENERAL PARALYSIS; SOME PSYCHOLOGICAL AND PHYSICAL OBSERVATIONS. G. DEM. RUDOLF, J. Ment. Sc. **71**:30 (Jan.) 1925.

Thirty-one cases are described after malarial inoculation and followed from two and one-half to fourteen months. An attempt has been made to quantitate delusions of grandeur: one patient estimated his high-jump record at three miles, but reduced this after seven months to ten feet. In eight cases, the pupil reactions changed for the better. In general, physical improvement occurred in every case and mental improvement only in the early cases.

BOND, Philadelphia.

EXPERIMENTAL STUDY IN MENINGEAL IRRITABILITY. MICHIO KASAHARA, Am. J. Dis. Child. **28**:407 (Oct.) 1924.

Using the number of cells per cubic millimeter in the spinal fluid as an index to the severity of meningeal irritation, the author observed the effects of simple aspiration, injections of sodium chlorid solution, varying from 0.1 per cent. to 4 per cent., Ringer's solution, human cerebrospinal fluid and fresh and inactive serum. All these experiments (including simple aspiration) resulted in an increase in the cellular constituents. Ringer's solution and sodium chlorid of 0.6 per cent. strength were found to be least irritating. Fresh serum was more irritating than inactive serum. Fresh serum from an animal of a different species was most irritating of all.

VONDERAHE, Cincinnati.

THE RELIGIOUS FACTOR IN MENTAL DISORDER. EDWIN R. EISLER, J. Abnorm. Psychol. **19**:85 (April-June) 1924.

While deprecating the supernatural as an element in the explanation of human conduct, the author acknowledges its almost universal presence and acceptance as a manifestation of divine power. The belief in God and in immortality can be considered as wish phantasies. Four cases are cited briefly. When a religious element dominates the psychotic picture, it is patterned on the patient's traditional faith. Religion can affect the child powerfully, excite fear of the unknown and initiate feelings of inferiority.

HAMILL, Chicago.

THE BASAL METABOLISM OF CHILDREN WITH IDIOPATHIC EPILEPSY. FRITZ B. TALBOT, MARY HENDRY and MARGARET MORIARTY, Am. J. Dis. Child. **28**:419 (Oct.) 1924.

A study of the basal metabolism in eleven epileptic children was undertaken because of the suggested use of thyroid extract in the treatment of this disorder. In all cases the basal metabolic rate was found to be normal or increased, leading to the conclusion that thyroid therapy is not indicated in epilepsy of childhood.

VONDERAHE, Cincinnati.

PREVALENT MISUNDERSTANDING CONCERNING UNCONSCIOUS MIND. TOM A. WILLIAMS, J. Abnorm. Psychol. **19**:77 (April-June) 1924.

The author expresses disapproval of the use by psychiatrists of the idea that subconscious or unconscious elements must be appealed to in the therapeutic explanation of the morbid phenomena. The article seems like a wee small voice very deep in the wilderness. The heart of the mystery is to be found by anamnestic skill as soon as the reluctances are done away with. The term "reluctance" appeals to the author more than repression.

HAMILL, Chicago.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 7, 1925

SHERMAN F. GILPIN, M.D., *President, in the Chair*

TORSION SPASM. DR. CLARENCE A. PATTEN.

This patient is presented as having a case of torsion spasm, which is interesting from the standpoint of the differential diagnosis between a lenticular disease or torsion spasm, a torticolis and a functional nervous condition.

A woman, aged 30, unmarried, a bookkeeper, had had frequent attacks of sore throat all her life. The family history was negative. The personal history was negative so far as birth and development are concerned. The patient was in school until 17 years of age, reaching the 9th grade. Her menses were established at 16, and had been regular; at the age of 7, she had an attack of chorea which lasted five weeks.

In 1915, she noticed an occasional twitching in her neck which persisted for some time and which was accompanied by pain in the right side of the neck, the back and on the top of her head. The scalp was tender to the touch. There were also quivering feelings in the back of her neck and in the viscera. This attack lasted one year. She recovered completely, except that there was a recurrence of trembling in the back of her neck and pain in her neck and shoulders when she became nervous. The second major attack occurred in 1919, following a period of rather hard work. The head was occasionally pulled to one side in a spasm of the neck muscles, and the head twitched at times. After from five to fifteen minutes, the muscles would relax and the head would resume a normal position. There was also pain in the back of the head and in the neck at this time. This attack lasted for about fifteen months, but altogether it was not as bad as the first attack. From this time until October, 1924, the patient had brief and rather abortive attacks of twitching of the head and pain in the neck, but at no time were the symptoms continuous. In October, 1924, the present attack began. Before the attack came on, the patient was having some difficulty with her manager, who was constantly complaining about her work and nagging her. She had been under the care of a physician continuously since 1919. The present trouble is characterized by twitchings in the neck and spasms in which the head is projected forward. The neck was more or less continuously stiff in the beginning and she was in bed for five weeks; during this time she could not get her head off the pillow. The head was not pulled over to one side as in the first attack but the occiput was pulled backward and the chin thrust forward, which condition has obtained up to the present time. The condition is always worse during her menstrual periods.

The physical examination revealed a rather suspicious condition of the teeth, the roentgenogram showing a possible abscess at the root of one of the molars. Four teeth had been removed for abscess at the roots. The abdomen was flat with some tenderness over the appendix. The liver was not palpable. There was a slight kyphosis and scoliosis to the right, probably postural.

The neurologic examination showed active reflexes, none being pathologic, however. With the exception of the spasms of the neck, face and back muscles, the neurologic examination was negative.

There is an irregular hypertonic spasm of the neck muscles in which the head and chin are thrust forward and the occiput drawn somewhat downward. During the spasm the face is contorted, the brow wrinkled and the lips pulled apart in a sort of half smile. At this time a fine tremor of many of the facial muscles and of the chin as a whole is noted. The erector spinae muscles enter into a state of spasm coincident with the contractions of the muscles of the neck and face. These movements last from a few seconds to a minute or more and are often initiated by attempts to talk or eat. The spasms are not always of the same intensity, as when the patient is undisturbed or lying in bed they are apparently not as severe as when she is making an effort or being observed. The spasm has not been observed during the time that she is sleeping, but there is a slight nodding movement. To quote Kinnier Wilson, there is a temporary hypertonus followed by a temporary hypotonus in a confused and irregular fashion. The tremor of the chin and muscles of the face is not constant but is nearly so, and is often observed when the muscles of the neck and back are not in a condition of spasm. Curiously enough, the torsion spasm is less marked when the eyes are closed. There is no demonstrable weakness of any muscles and no tenderness except possibly a slight sensitiveness over the insertion of both trapezii. At times tremors of moderate intensity are observed in the muscles of the chest and back. When not in the spasm it is noted that the patient winks frequently, and often when she is lying relaxed it is noted that there is a slight and more or less constant projection motion of her head. No athetotic movements have been observed. The patient has improved somewhat under rest in bed and symptomatic treatment.

Electrical examination of the muscles of the neck and back were negative except for a possible increased irritability. Roentgen-ray examination of the cervical vertebrae was negative, and there was no nerve tenderness. Roentgen-ray examination of the sella turcica revealed no pathologic condition, but it measured 4 mm. by 1 cm. The urinalysis, blood count, blood chemistry and other laboratory tests have been negative.

In the absence of definite arthritic changes and tenderness of muscles or nerve trunks, a torticollis of toxic infectious origin can probably be ruled out. Furthermore, the involvement of the muscles of the back shows that the spasms are not confined entirely to the neck. A diagnosis of functional nervous disease is hardly tenable in the presence of the tremors and slight movements of the patient's head during sleep. I believe that this is a case of torsion spasm with periods of remission, beginning about ten years ago. Many writers on the subject of lenticular disease have mentioned the fact that remissions are likely to occur. It is doubtful whether in any of the so-called remissions the patient has ever been entirely free from symptoms. The picture presented by this patient illustrates the postulates laid down by Wilson concerning the symptomology of lenticular disease; that is, variability of muscle tone, the appearance of involuntary movements and the absence of paralysis.

DISCUSSION

DR. F. M. R. WALSHE, London, England: In making a diagnosis of pseudosclerosis we at once encounter the difficulty that the series of conditions for which Westphal and Strümpell originally employed this term in the

last century bear no discoverable relation, clinical or pathologic, to the malady for which the term is now used by German writers. This has been pointed out by Kinnier Wilson, who has aptly observed that the Westphal-Strümpell cases form a collection of incompletely elucidated and obscure conditions and do not represent either a clinical or a pathologic entity. The malady now known as pseudosclerosis is no other than the progressive lenticular degeneration first adequately described by Wilson in 1912, and the criteria hitherto adopted to differentiate a malady, pseudosclerosis, from a distinct malady, progressive lenticular degeneration, are now recognized to be wholly illusory. For example, the corneal pigmentation once regarded as diagnostic of pseudosclerosis has now been reported in unequivocal cases of Wilson's disease. Similarly, the widespread cerebral lesions without gross macroscopic changes in the lenticular nuclei formerly considered to differentiate the former from the latter are now known to occur in progressive lenticular degeneration. In other words, this malady is not the pure corpus striatum system disease it was originally believed to be. It was inevitable that with accumulating clinical and pathologic observations, the original clear-cut conception we owe to Wilson must ultimately come to be broadened so as to include a range of clinical and pathologic variations such as are found in every malady of the nervous system. This in fact has happened, and there is no longer any justification for believing in a separate disease "pseudosclerosis" or for retaining in neurologic literature a term so vague and misleading, and one which serves no other pupose than to confuse thought and to confer a wholly undeserved priority on Westphal and Strümpell. With regard to Dr. Patten's case of torticollis, Foerster's suggestion that such a condition may be the incomplete expression of a corpus striatum lesion is interesting. It has to be remembered that in the erect human subject the carriage of the head is a complex piece of coordination, on which the whole postural regulation of the musculature in a measure depends. This being so, it would not be surprising if, in accordance with Hughlings Jackson's notions of dissolution of function in the nervous system, disorders of this function were an early expression of disease in the underlying nervous mechanisms. However, whether or not this mechanism is situated in the basal ganglia remains wholly a matter of speculation.

DR. PATTEN: It is my belief that the greatest argument in favor of a possible lenticular disease in many of the cases diagnosed as torticollis is that the patients do not recover from the spasmodic condition of the neck. In almost all other conditions in which there is a physical, that is a toxic-infectious basis for the symptoms, it is possible to influence the disease by proper therapy. The history of the usual cases of torticollis is that the condition continues until the death of the patient.

A CASE OF METASTATIC CARCINOMA IN THE SPINAL MENINGES FROM A PRIMARY GROWTH IN THE PROSTATE. DR. J. HENDRIE LLOYD.

A colored man, aged 49, suddenly developed symptoms of a transverse lesion in the lower spinal cord. There was a rapidly advancing paraplegia with retention and anesthesia (partial or complete) below the lower costal margins; also an acute bed sore. For about a year, the man had had symptoms of prostatic enlargement, such as dysuria and dribbling; and more recently there had been great difficulty in passing the catheter. A surgical examination revealed an enlarged prostate gland of "suspicious" character. The patient died a few weeks after the onset of the spinal symptoms. Necropsy revealed

primary carcinoma of the prostate gland, with metastatic growths in the dura of the lower dorsal and the lumbar regions, as well as in the lung, peribronchial lymph gland and capsule of the liver.

Dr. R. B. Richardson demonstrated the sections on the screen.

DISCUSSION

DR. WILLIAM G. SPILLER: The case presented by Dr. Lloyd has similar features to the case reported by me before this society in October, 1924. When I presented my case I was not aware of any case like it in the literature. My paper appeared in the April issue of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY.

DR. LLOYD: The onset of the cord symptoms, acording to this patient's own statement, was abrupt; but there can be little doubt on looking at the specimens that he must have had some preliminary symptoms. The roentgen ray had shown involvement of the bones of the spine, pelvis and femora; and Ewing in his work on tumors says that metastatic growths, of a markedly osteoplastic kind, are rather common in carcinoma of the prostate gland. In the present case the bony tissue had not been examined under the microscope, but the slides from the other growths had been seen by Dr. Goforth and Professor Allen Smith, who pronounced the tissue carcinoma.

THE RELATIONSHIP OF THE RADIX MESENCEPHALICA TRIGEMINI TO THE EXTRA-OCULAR MUSCLES. DR. WALTER FREEMAN.

The location of the sensory ganglia of the nerves supplying the extra-ocular muscles has been one of the problems with which anatomists have long been confronted. These nerves are described as purely motor, and yet it has been shown that they contain centripetal fibers. There are no dorsal root ganglia, although occasionally some ganglion cells are found scattered among the fibers of the nerves. The sensory nerve endings in the muscles are said to be extremely numerous, although true muscle spindles, according to Piersol, are absent. The scattered nervous elements occasionally found are insufficient to account for the rich sensory supply of these muscles.

I have been led to believe that the radix mesencephalica trigemini contains the sensory ganglion cells which furnish muscle sensibility to these extra-ocular muscles.

The radix mesencephalica trigemini has been the object of numerous investigations since its discovery by Deiters in 1865 (Untersuchungen über Gehirn und Ruckenmark des Menschen und der Sangetiere, Braunschweig, 1865, pp. 91 and 92). This author immediately noticed the resemblance of the cells to those of the spinal ganglia, but no other instance of intracerebral sensory ganglion cells was known at this time, so he assigned to them a different function. Later studies in comparative anatomy showed that in the lower fishes there are no dorsal spinal ganglia, but instead a column of large, rounded, unipolar cells situated dorsolaterally to the spinal canal. In 1909, Johnston (J. Comp. Neurol. **19**:593, 1909) recognized the analogy between the radix mesencephalica trigemini and these Rohon-Beard cells, and stated his belief that these cells furnished muscle sensibility to the muscles of mastication. This opinion has been accepted practically universally.

The cells are disposed along the course of the root in an uneven manner. There is a large group above the entrance of the trigeminal nerve, but then the cells practically disappear until the level of the trochlear nucleus is reached where another group is found. The root again becomes attenuated,

but enlarges considerably in the vicinity of the oculomotor nucleus and continues up to the level of the posterior commissure.

The reason for this long forward extension has not been understood. It has been shown that the third division claims by far the majority of the fibers, the second and first divisions taking only a very few. It would be expected that the ophthalmic ramus, which belongs to the ophthalmic neuromere, would contain many fibers coming from the upper part of the tract, and yet this is not the case. Section of the ophthalmic ramus is followed by practically no degeneration in the radix mesencephalica trigemini.

Section of the central root of the gasserian ganglion is followed by degeneration of a large number of cells in the radix, but this degeneration is by no means complete; in the cephalic portion of the root there are especially many well preserved cells and fibers. The degeneration is limited to the side of operation. Therefore there must be some other outlet for the fibers of these cells.

Golgi (Arch. ital. de biol. **19**:1893) gave the first intimation that these cells were connected with the extra-ocular muscles. In 1893, he described fibers coming from the large globoid cells of the radix mesencephalica trigemini and joining the trochlear nerve. He believed that the cells were of motor type. On account of the difficulty in impregnating these cells satisfactorily, his results were not confirmed, and Cajal denied any connection with the trochlear nerve.

A clinical case reported by Van Valkenburg (Folia neurobiol. **5**:360, 1911) throws light on the subject, although his interpretation is debatable. He studied a case of intracranial tumor pressing on and destroying the ophthalmic ramus of the trigeminal nerve. In this case he found profound degeneration in the mesencephalic root in its upper portion, and made the deduction that the fibers from these cells traveled in the ophthalmic ramus. This is the only instance known, and is in direct contradiction with the results of many experiments. In this case, however, there was complete ophthalmoplegia. I suggested (Folia neuropathol. Esthoniana **3**:27, 1925) that the paralysis of the oculomotor nerve was the cause of the degeneration of the radix mesencephalica trigemini in its upper levels.

In order to test out this hypothesis, a series of experiments was undertaken. In four cats the orbit on one side was completely exenterated, and in another animal, used as a control, the eyeball was enucleated. After a lapse of time the animals were killed, and the brain stems cut in serial sections. One was subjected to the Marchi technic, the others to cell stains.

It was found by the Marchi method that there was slight degeneration in the radix mesencephalica trigemini on the side of the lesion at the level of the third nerve and on the opposite side at the level of the fourth nerve. The nerves showed slight but definite degeneration.

In the brain stems studied by means of cell stains, it was found that many of the large globoid cells either had disappeared completely or had become shrunken, deeply stained and distorted. They were obviously degenerated. The degeneration affected the upper portion of the root on the side of operation, although below the level of the oculomotor nucleus the degeneration was more marked on the opposite side, that is, on the side of origin of the trochlear nerve. There was no degeneration in either of the two roots below the trochlear level. The oculomotor nucleus on the side of operation was markedly degenerated, and some chromatolytic changes were noted on the opposite side. The trochlear nucleus was practically completely degenerated on the opposite side.

The animal whose eyeball had been removed showed no degeneration of the radix mesencephalica on either side.

The results of the experiments, in conjunction with other data given here, strongly suggest that the radix mesencephalica trigemini supplies muscle sensibility to the extra-ocular muscles.

DISCUSSION

DR. N. W. WINKELMAN: I should like to ask Dr. Freeman whether he differentiated between the group of cells that is scattered along this root, the so-called intertrigeminal nucleus of Kohnstamm, and the cells of the mesencephalic root of the fifth? How would Dr. Freeman explain the fact that after extirpation of the eye muscles by other authors they could find no cell atrophy or fiber change in the mesencephalic root?

This was done by Kohnstamm and Quensel in 1908, and by May and Horsley in 1910, in carefully controlled experiments.

DR. FREEMAN: I noted some smaller cells along the course of the radix mesencephalica trigemini. They were often difficult to differentiate from the atypical globoid cells, even though care was taken to avoid shrinkage by double embedding. The small cells presented no obvious deviation from the normal. I might suggest that I regard these cells as homologous with those of the column of Clarke in the spinal cord, but I have not had an opportunity to verify this hypothesis. The paper by May and Horsley dealt with section of the radix mesencephalica trigemini intracerebrally, but I remember no reference to operations on the extra-ocular muscles. (In rereading the paper I find as a conclusion that they state that the radix mesencephalica has nothing to do with the musculature of the palate or with the extra-ocular muscles, but there is no mention of any experiments in this connection.) These investigators reported that there was no loss of power in the muscles of mastication.

SPINAL CORD TWENTY-FOUR YEARS AFTER MYELORRHAPY FOR COMPLETE DIVISION. DR. A. E. TAFT and DR. J. P. PAUL (by invitation).

The patient, in 1901, at the age of 26, was admitted to the Pennsylvania Hospital with a gunshot wound in the back. The bullet had entered 1 inch (2.5 cm.) to the right of the seventh dorsal spine and had passed directly into the spinal canal. There was immediate complete motor and sensory paraylsis below the level of the tenth dorsal spine, and all superficial and deep reflexes were abolished. Operation was performed by Dr. F. C. Stewart three hours after the injury. The membranes were lacerated and the spinal cord was completely severed. The divided ends of the cord were approximated with catgut sutures, and the wound was closed. Surgical recovery was uneventful. At the time it was thought that there was a "partial return of function in the lower limbs" (Stewart and Harte: Philadelphia M. J., June 7, 1902).

Dr. Cadwalader examined the case in 1919 (Ann. Surg., June, 1920) and found complete paralysis of motion and sensation below the level of the tenth thoracic spine. The lower limbs felt rigid, but there was no wasting. Involuntary flexor movements of the lower limbs occurred at irregular intervals and were increased by stimulation. Extreme rigidity of the lower limbs was present and prevented the tendon reflex responses. A bilateral Babinski sign was present, and the abdominal reflexes were absent. The bladder was emptied automatically. The patient's general health was good.

The necropsy (partial), performed by Dr. Paul, revealed septicemia, bronchopneumonia and massive decubitus over the sacrum. The dorsal and lumbar portions of the cord were removed for detailed study.

About midway the length of the specimen, palpation suggested that nerve tissue was almost entirely, if not wholly, absent for about 3 cm. Above this point the cord was much shrunken in all its diameters, in particular anteroposteriorly. Below, there was only moderate shrinkage, and the lumbar enlargement showed relatively little change in size. The spinal nerve roots related to the thinned portion were like small threads. The dura was adherent to the underlying membranes for a distance of about 1.5 cm. over the thinned-out area. On dissecting this away, the pia-arachnoid appeared thickened and fibrous, with small round nodules of cartilaginous consistence in the tissue.

Microscopic sections above the lesion, which represent the dorsal cord, stained by the Weigert method, showed the pia thickened. There was lack of staining in both columns of Goll, to a slight degree in the inner sides of the columns of Burdach, only scattered staining in the lateral and anterior pyramidal tracts and marked lack of staining throughout the entire periphery.

The sections from the cord below the lesion showed only lateral column change. This appeared as a triangular unstained area in the periphery of the cord, with a narrow tongue extending posteriorly as far as the posterior horn. In the sciatic nerves, some of the fibers stained deeply as in normal tissue, while others were only tinged by the stain. In cross section, however, the myelin sheaths of all fibers appeared to be of average thickness.

The sections from the area of the lesion itself contained no nerve fibers. There was only a very fibrous dura within which were areas of bone formation containing islands of bone marrow; a somewhat thickened pia; on one side, a small fragment of very cellular glia tissue and the fibrous septums of the cord bearing many blood vessels.

The gray matter of the cord above and below the lesion was not notable. The cell stain showed that the nerve cells were present in the anterior horns, and they contained well stained Nissl substance. The cells of Clarke's column were also present and normal.

The neuroglia stain revealed a diffuse increase of glia—both cellular and fibrillar—present in all sections, but much greater in degree above the lesion.

DISCUSSION

DR. WILLIAM B. CADWALADER: This case is unique because of the long survival, twenty-four years, after complete section of the cord. The patient presented the characteristic signs of transection of the spinal cord. At first there was typical spinal shock. The lower limbs were flaccid and the tendon reflexes were absent. It is recorded that on the twenty-first day one patellar tendon reflex appeared and gradually other reflexes returned, the limbs became spastic, and finally the typical picture of the so-called second stage, i. e., of "mass reflex" as described by Riddock (Brain **40**:224, 1917) was established. This continued without variation until a month before presentation, when a bed sore of the buttocks followed by fatal septicemia developed. The hypertonicity of the limbs presented interesting features for analysis and discussion. In 1902, Dr. Stewart believed that there was actual return of voluntary motion, but at that time the significance of the spinal reflexes appearing after having been abolished by spinal shock was not understood, and consequently misinterpreted. During my examinations in 1920 and subsequently, but before she

became infected, it was found that the lower limbs did not always assume the same degree of flexion. But if placed by the examiner in a partially flexed posture, the limbs would remain so until some stimulus produced an alteration reflexly. When the patient was lying on a bed, the legs could be fully extended by the examiner, overcoming the spasticity of the flexor muscles by gentle force, and then they would remain extended a few moments until stimulation produced an alteration of posture by a reflex flexor response. I must admit that I am not entirely clear as to the interpretation of these phenomena, but I am inclined to the opinion that they are evidences of the existence of plastic tone in the extensor muscles of the lower limbs. According to Riddock, who quotes Sherrington, "reflex tonus" or "plastic tonus" of Langehaan does occur in the extensor muscles of the spinal dog, it occurs rarely or not at all in the spinal monkey, and it seems to disappear entirely in spinal man. If the evidence is sufficient to establish the fact that plastic tonus was present in this patient, it probably can be explained by the fact that reflex phenomena, having existed so many years, became unusually well developed. Similar cases of long survival may be recorded later, since some of the patients who had this condition during the war may be living.

DR. WALSHE: Was there any residual urine after evacuation?

DR. CADWALADER: It is not positively known, but I assume that there was residual urine. The patient had not been catheterized for years.

DR. WALSHE: There is always some amount of residual urine, and what happens is not true reflex micturition but a mutilated reaction in which relaxation of the sphincter plays a more important part than contraction of the bladder. I think automatic micturition in the strict sense of the term never occurs in spinal animals or in such spinal transections in man as have come under my observation.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Feb. 19, 1925

HERMAN M. ADLER, M.D., *President, in the Chair*

A CASE OF OCCLUSION OF THE POSTERIOR-INFERIOR CEREBELLAR ARTERY. DR. G. W. HALL.

Mrs. B., aged 48, was admitted to the hospital, Feb. 2, 1923, with a history of having had occasional morning headaches in the occipital region from July, 1922, until Dec. 14, 1922, when they suddenly became very severe and were definitely located on the left side. She complained of inability to swallow and to raise the eyelids normally, severe vertigo and some blurring of vision. All these symptoms disappeared within twenty-four hours, and she felt well again. On Jan. 24, 1923, she suffered a second attack of the same symptoms. In addition, she was troubled with nausea when lying on the left side and also complained of disturbance of sensation in the right arm and leg; she was unable to detect heat or cold. At this time, she also had marked polyuria lasting several days. During the period from July, 1922, to Feb. 13, 1923, she had a blood pressure that ranged from 130 systolic and 90 diastolic between the attacks to 190 systolic and 130 diastolic during the attacks; at the same time her pulse rate was 52, and the rhythm was markedly intermittent.

Examination on Feb. 13, 1923, revealed a definite Horner's syndrome of the left eye, slight dulness of sensation on the left side of the face, and slight weakness of the seventh nerve on the left side. There was no deafness or ringing in either ear. There was slight ataxia of the left arm in the "finger-to-nose" test; the lower extremities showed nothing abnormal, and there was no Babinski sign on either side. The gait showed a constant tendency to fall to the left. There was diminished sensibility to pain and temperature over the right arm and leg, but not over the right side of the face. There was no dissociation of temperature sense.

The patient was placed on a modified diet and potassium iodid was administered occasionally; she has had no attack since Jan. 24, 1923.

A Case Presenting the Cestan-Chenais Syndrome. Dr. John Favill.

A man, aged 38, a solderer, was admitted to the Cook County Hospital a week before presentation, with a history of having been stricken with sudden paralysis of the right arm and leg while at work. By the following day, there was only a slightly increased knee reflex and a Babinski sign on the right. When admitted to the hospital, there was apparently some dilatation of the right pupil, but this was really the Horner syndrome with contraction of the left pupil. There was weakness of the left side of the palate, with difficulty in swallowing. The man could make no sound at first, but speech had improved markedly. There were diminution of pain and temperature sense on the right side of the body and face, a slight tendency to deviate to the left in walking, and some incoordination of the left arm.

The symptoms pointed to a lesion of part of the left posterior-inferior cerebellar, or a near-by branch of the vertebral artery. The case belongs to the Cestan-Chenais group. The Wassermann reaction had not been completed.

DISCUSSION OF CASES OF DRS. HALL AND FAVILL

Dr. Loyal Davis: Clinical cases of lesions of the posterior-inferior cerebellar artery have stimulated experimental work on the spinal tract of the fifth cranial nerve. Dr. Margaret Gerrard, as the result of experimental work on the size, course and distribution of the fibers entering this tract, a review of practically all published cases of involvement of this artery, and a correlation of the clinical findings with histologic facts, concluded that the fibers of the spinal tract bifurcate after entering the pons; some of the fibers go centrad and others course downward into the spinal cord. She believed that the fibers which transmit pain and temperature sensations also transmit touch impulses, but predicated that the fibers were attuned differently when these two types of impulses passed over them.

Within the past few months Windle, working in the Department of Anatomy of the Northwestern University Medical School, has shown by accurate staining methods that, in addition to the fibers described by Gerrard, there are very fine fibers which come into the pons and immediately turn downward without bifurcation. He was able to trace these fibers directly into the spinal nucleus of the trigeminal nerve, and states that they lie on the periphery of the bundle of descending fibers. He is inclined to believe that the latter fibers carry pain and temperature impulses, while the former fibers, first described by Cajal, transmit sensations of touch. This is of course in keeping with the fact that the spinal tract of the fifth nerve is in all probability a direct upward continuation of the substantia gelatinosa rolandi, which is so intimately concerned with the fibers carrying the same type of impulses within the spinal cord.

It is not common to find loss of sensation on both sides of the face in a lesion of the posterior-inferior cerebellar artery. It suggests the involvement of other arterial branches, or, if a circumscribed occlusion of that artery exists, the secondary neurons of the fifth nerve are involved.

SPINA BIFIDA OCCULTA CERVICALIS. DR. G. B. HASSIN.

This paper will be published in full in a later issue of the ARCHIVES.

RESPIRATORY ARRHYTHMIA AND HYPERPNEA. SIGMUND KRUMHOLZ.

A Jewish girl, aged 24, was admitted to the Cook County Hospital, Nov. 8, 1924. The outstanding symptom of which she complained was marked difficulty in breathing; this was accompanied by considerable weakness (she had lost 30 pounds [13.6 kg.] in weight) and insomnia. The symptoms had set in suddenly about six months before admission and had persisted. The respiratory disturbance was most distressing, but for short intervals she was fairly comfortable. Breathing was normal during sleep. About six months before the onset of this condition, the patient had an attack of "sleeping sickness." Lethargy was marked for two or three days and was accompanied by intense headache and diplopia. Then it continued for three weeks longer in a milder form, when she was drowsy and periodically fell asleep while standing or sitting.

About 1917, her mother was shot dead in front of her, and her younger brother, who was left in her care, died in 1919. Prior to the encephalitic attack, she worried about a love affair, which she insisted was only a passing episode.

Examination revealed marked emaciation and disturbed breathing. The respiratory disturbance had several features. It was irregular, jerky, rapid, noisy, increased in force and rate, and yet on diversion of attention, it became practically normal. During the forced respiration, the accessory muscles were prominent. On several occasions, I observed periods of apnea during which her lips were definitely cyanosed. This condition was invariably followed by a complaint of marked distress and a return to the hyperpnea. On account of the irregularity, it was difficult to count the respirations, but roughly the rate varied between 35 and 50 per minute. The pulse was weak and ranged between 85 and 100; the temperature was 98 F., and the blood pressure was 95 systolic and 60 diastolic. The basal metabolism rate on February 16 was plus 26 per cent. The neurologic examination was essentially negative; deep reflexes were markedly increased, and there was an external strabismus of the right eye.

As this patient presented no signs of an organic lesion, a hysterical condition was considered. The negative findings, the frequent absence of respiratory disturbance during diversion of attention, and the fact that the patient experienced several emotional shocks some time prior to the onset of the disease, might offer justification for such a diagnosis. The patient presented no stigmas, however, and there was no response to therapeutic interference. The persistency of the respiratory disturbance for seven months so that the general health of the patient was seriously undermined suggested that the condition was produced by a lesion in the central nervous system, most probably in the respiratory center of the medulla, possibly in the controlling centers higher up. I believe that this syndrome is a sequel to lethargic encephalitis.

DISCUSSION

DR. JULIUS GRINKER: Has hypnotism been tried in this case? If not, I think it would be advisable to try it.

DR. LEWIS J. POLLOCK: This case is of interest from the physiologic standpoint for two reasons: first, no evidence of tetany has ever been detected, although the patient for many hours is in a state of hyperpnea. This is particularly interesting because of the marked ease with which tetany has been produced in athletes by forced respiration. Second, it is of interest as regards the possible location of the lesion producing the respiratory symptoms. Because of its adjacence to the midbrain, it is possible that the lesion is situated in the superior colliculus. In the course of experimental work, Dr. Loyal Davis and I produced marked hyperpnea by the intravenous injection of picrotoxin; a normal rate of respiration then followed decerebration below the colliculi. I see no reason why this case is not one of organic disease.

DR. SIGMUND KRUMHOLZ: Suggestive therapy was employed, but I do not think that hypnotism will be of any avail. I have considered carefully the possibility of hysteria in the case; I did not undertake a thorough psychanalysis of this patient, but I have taken a careful history of her experiences. She suffered several severe psychic traumas when younger, but she had had no previous hysterical attack. I have concluded that this is not a psychogenic case. In attacks of apnea, she may become markedly cyanosed, even during a period of diversion. In these attacks it almost appeared as though she had forgotten to breathe. On one such occasion, I had to arouse her to resume breathing. In this attack, I could detect no external cause for the apnea except absent-mindedness while listening to a conversation between her brother and myself. Even though the hyperpnea may be functional in origin, I do not think the attacks of apnea are produced by hysteria.

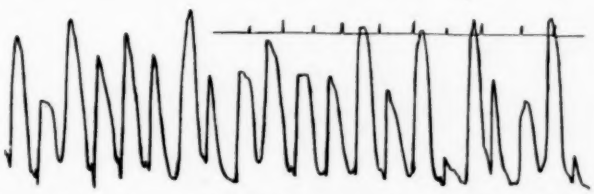
A CASE OF DIABETES INSIPIDUS DUE TO SYPHILIS. J. E. GORDON and PETER BASSOE.

A salesman, aged 39, rather obese but of healthy appearance, developed symptoms of severe diabetes insipidus in September, 1924. A positive Wassermann test was obtained with the spinal fluid, and after the institution of antisyphilitic treatment, his general strength improved but the polyuria persisted. Detailed metabolic studies were made, and the effect of pituitary extract as well as of diet on the diuresis was striking. These were the main features: (1) Pituitary extract given in doses of 1 c.c. daily caused concentration of urine from an average volume of 8,000 c.c. per day to as little as 3,000 c.c. (40 per cent. of control volume) per day, with no change in the total output of nitrogen, chlorids or sugar. (2) A high salt diet gave rise to markedly increased diuresis of approximately 70 per cent. with no change in the output of nitrogen or glucose, but with greatly increased chlorid output. (3) A high carbohydrate diet relieved the symptoms of thirst and burning of the mouth, and may have had a depressant effect on uric acid metabolism. (4) A low protein, low salt diet was efficient in diminishing the intake of fluids, and likewise decreased the output of urine which regularly parallels the intake. (5) A high protein diet served to increase to some extent the total output of fluids. (6) The general conclusions from dietary management indicated that the best results were obtained with low protein, salt-free diets. (7) The uric acid of the blood was consistently at a high level, irrespective of dietary management or the administration of pituitary extract. (8) Following spinal

puncture, there was an increased urinary output, which exceeded previous levels despite daily injections of pituitary extract. This effect persisted for three days, after which the concentrating power of pituitary extract was again evidenced. (9) There was no evidence of impaired kidney function, except possibly increased uric acid values. The phenolsulphonephthalein test was within normal limits. (10) The essential pathologic condition seemed to be a syphilitic process involving the region about the hypophysis. The spinal fluid Wassermann reaction was ++; the Lange test was positive; the cell count was 9. (11) Roentgen-ray examination revealed no definite change about the sella turcica.

A Case of Postencephalitic Hyperpnea. J. E. Gordon and Peter Bassoe.

A woman, aged 31, married, first admitted to the Presbyterian Hospital in October, 1923, had been well until January, 1921, when she had an acute illness with fever, delirium and diplopia. The diplopia lasted about four weeks, and she was very drowsy during that time. After the subsidence of these symptoms, she failed to regain strength and appetite, but had no active subjective symptoms until about a year later, when she had an attack of what was called mucous colitis, and she suddenly developed attacks of rapid and forced breathing.



Kymograph tracing of respiratory movements during hyperpnea.

These seemed to be preceded by a sensation of pressure along the descending colon and occasionally in the epigastrium. Weight decreased from 151 pounds (68.5 kg.) to 97 pounds (44 kg.) in the course of six months. The attacks were brought on whenever the patient was questioned or irritated in any way. She had a worried expression and appeared to be extremely sensitive; the condition might easily have been mistaken for hysteria. A general neurologic examination at that time was negative. A basal metabolic rate determination was attempted, but was unsatisfactory on account of the forced breathing. The rate obtained was plus 68. Her weight at that time was 95 pounds (43.1 kg.). The carbon dioxid of the blood was 32.4 volumes per cent., and the hydrogen-ion concentration (p_H) 7.7. During her stay of one month in the hospital, she had many hours of forced breathing every day. She was weak, restless, irritable, uncomfortable, and ate poorly, but gained 5 pounds (2.3 kg.).

The patient returned to the hospital, Jan. 3, 1925, when she weighed 104 pounds (47.2 kg.) and still complained of abdominal distress, but the attacks of forced breathing were less frequent. She had learned to suppress her respirations, but thought she did this at the cost of increased abdominal discomfort. The attacks had gradually decreased in frequency during the past month. On January 9, the basal metabolic rate was minus 8.

The attacks of hyperpnea at the present time are brief and paroxysmal, rather than long continued. Periods of normal breathing are interspersed with occasional outbursts of abnormal respirations. The effort in the latter instance

is loud and labored, suggesting the panting of a dog. Without apparent reason, the breathing suddenly becomes deeper and the rate more rapid, averaging 40 to 45 per minute. Inspirations are long and deep, expirations labored. The mental attitude during the paroxysms is characteristic. The face is drawn, the patient is in apparent distress, and has an anxious expression. She becomes definitely restless, and the whole musculature is tense and rigid. The attacks last from five to fifteen minutes and terminate as abruptly as they begin. At no time have we observed a crescendo-like increase in the rate of respirations as in Cheyne-Stokes breathing.

In kymographic tracings (shown in the accompanying figure), made during one of the attacks of hyperpneic breathing, the respirations are seen to involve a forced effort with each inspiration and expiration. No definite rhythm can be discerned, although analysis of the record indicates that usually there is a 2:1 ratio; two deep respirations are followed by a subsequent maximal effort. In some instances, this ratio may be three to one; and occasionally, only one shorter or less forced respiration intervenes between the maximal effort.

DISCUSSION

DR. PETER BASSOE: Our case illustrates some of the points spoken of by Dr. Krumholz. There are no other signs of a postencephalitic state. There are in the literature a number of cases of hyperpnea without the parkinsonian syndrome, and all have the same symptoms. The patients have lost weight rapidly, and all are uncomfortable. Pardee, of New York, comments on this, and accounts for the condition by the supposed termination of the autonomic system in the thalamus, rather than by a bulbar lesion. There seem to be many cases just like the one presented. In all, the patients have had encephalitis, and it is far-fetched to think that there is not a direct connection of cause and effect, even if many of them do show a certain neuropathic tendency. It may be that it is necessary to assume a neuropathic basis, for of the many thousands of cases of encephalitis, comparatively few are followed by hyperpnea.

In the case of diabetes insipidus presented, although we can be certain in this case that it is due to syphilis, it is important to treat the patient by regulation of diet and administration of pituitary extract, because if we do not, he may perish before we have a chance to cure the syphilis. I wish to emphasize how desperately ill this man really was before he came to the hospital. He could hardly drag himself into my office. He was given antisyphilitic treatment by his own physician for about six weeks, and improved in strength; but the polyuria persisted, and it was only after these other measures were added that the decided improvement began. Now, by continuing the administration of pituitary extract and the regulation of diet, we shall probably be able to keep the patient in a satisfactory condition until the antisyphilitic treatment has produced its effects.

DR. RALPH C. HAMILL: During the epidemic, I saw several myoclonic cases in which the myoclonus was either synchronous with or double the respiratory rate; as the myoclonus was of the accessory muscles it seems that in those cases there was a definite relationship between the rate of respiration and the action of the accessory muscles. I think there must be some lower centers in which the control of these accessory respiratory muscles is situated.

DR. JULIUS GRINKER: Without attempting to explain hyperpnea following epidemic encephalitis, and without going into the probable localization, I should say that it is our duty to differentiate postencephalitic hyperpnea from

that which occurs in hysteria. I saw similar cases of hyperpnea before encephalitis was reported in this country, which were correctly diagnosed hysteria. In one case presented this evening, there was rapid respiration, which was not spectacular or noisy; in the other case, there was rapid, rasping, noisy respiration, which if continuous would have made the patient a skeleton instead of a fairly well nourished girl. Does Dr. Bassoe or Dr. Krumholz claim that there is no hysterical hyperpnea, but only the type due to encephalitis? Is it possible to differentiate postencephalitic hyperpnea from hysterical hyperpnea?

Dr. George W. Hall: Dr. Bassoe has mentioned the fact that they treated the patient for the diabetes insipidus in order to obtain better results in the treatment of the syphilis. I am wondering whether or not improvement of the latter condition will in any way benefit the diabetes insipidus. I do not believe it will.

Dr. Gordon: The result of the antisyphilitic treatment, of course, remains to be proved.

Dr. Bassoe: We are using the diet and pituitary extract until the antisyphilitic treatment has time to have the effect we hope it will have. We are dealing here, evidently, with a fairly active meningo-vascular condition. There is an increased cell count, with positive globulin and Wassermann reactions. The activity of the process has subsided, but it remains to be seen how much destruction has actually taken place. If we accept the more modern view of the purely cerebral site of these centers, then I should say that it is a little more probable that the antisyphilitic treatment will be effective. We can imagine that there then will be a better result than if the posterior lobe of the hypophysis has been largely destroyed by arterial disease, with infarction and necrosis, as we would have to suppose according to the older theories. A Norwegian, K. Motzfeldt, has recently written an interesting book on diabetes insipidus. He tries to maintain a dual attitude and claims an interrelationship between the hypophysis and certain cerebral centers that are responsible for this syndrome. He does not believe in any exclusively hypophyseal or cerebral theory.

In regard to the differentiation Dr. Grinker mentioned, our patient at one time was just as noisy in her attacks as the patient Dr. Krumholz presented. All these cases are much alike. This woman has had attacks of hyperpnea for three years with decreasing frequency; a monosymptomatic hysteria with that course would be very unusual. There have been no other hysterical attacks of any kind. The patient is nervous, hypersensitive, inclined to worry and so on, but I regard that not so much as evidence of hysteria as of a neuropathic constitution. Given a history of encephalitis with hyperpnea as the only sequel, the diagnosis seems clear to me.

Three Months of Amnesia Following "Moonshine Whisky" Debauch. Dr. Barnet L. Lemchen (by invitation).

On Feb. 1, 1922, at the Chicago Medical Society, I pointed out that the psychosis following the use of "moonshine whisky" differs from that after ordinary alcohol in that the delirium does not take the form of delirium tremens. In "moonshine" delirium, the patients lie quietly, and when they recover know nothing of any hallucinosis during the delirium. Or the delirium may take the form of an automatism; the patient may perform almost any act, and on recovery remember nothing he did or said during the state of automatism. If an acute hallucinosis develops, it is usually of visual instead of auditory type, such as is common in ordinary alcoholism.

The patient presented has passed through an attack resembling a Korsakoff psychosis, differing, however, in that the patient has completely recovered with good insight. Recovery was rapid, almost sudden, and the patient remembers nothing that transpired during a period of more than three months. The patient, aged 56, was admitted to the Chicago State Hospital, Oct. 9, 1924. She had been drinking heavily for some time and often drank moonshine. About two months before admission to the hospital, while riding in a street car, she suddenly straightened out, appeared rigid and had to be almost carried home. After that her left side seemed paralyzed for a day or two, and she has not walked well since. She talked incoherently for some time afterward and has since talked irrationally at times, saying that she could hear a brother who has been dead for twelve years talking to her; she has also spoken of seeing visions. When admitted, she was disoriented and speech was rambling. General examination revealed little of importance. The patient could not stand alone, owing to weakness, and apparently had bilateral foot drop. The pupils reacted sluggishly but were equal and regular; articulation was normal; fine tremor was present in the tongue and fingers; the superficial reflexes were normal, the deep reflexes exaggerated. Spinal fluid and serologic examinations were negative.

Mentally, on October 11, the patient was quiet and orderly and was oriented for place and person. She gave details of her life correctly. Regarding her commitment, she said she was walking along the street looking at cars, when a friend came and took her teeth away from her. She then ran and passed two ruffians who chased her. She saw the sign of the hospital and came in (she was regularly committed from the Psychopathic Hospital). She said that her husband is in the penitentiary because he shot the Indiana County horse doctor; her husband drinks and shot her twice; her husband "holds up" people but never gets any money. She thought she had been in the hospital for two weeks when she had been there only two days. At times she talked to imaginary people, but denied that she saw or heard anything. The patient was in bed for almost three months, unable to walk on account of foot drop. She also developed anesthesia of the lower extremities. About the middle of January, 1925, she suddenly began to take notice of her surroundings. She required assistance in walking until Jan. 24, 1925, when she became clear and the foot drop improved sufficiently for her to walk unaided.

Mental examination, Jan. 24, 1925, showed that the patient was clear and oriented, but completely amnesic for the preceding three months. The last thing she remembered was getting dinner at home; her next memory was when she came to herself early in January. She remembered nothing of any hallucinatory experience. On the day she lost herself, she took three or four drinks. She realized that there must have been something wrong with her mind, for she was "just dead for several weeks."

DISCUSSION

DR. CLARENCE A. NEYMANN: In what way does this case differ from the ordinary Korssakow syndrome? The patient seems to have had all the classical symptoms of this disease. I do not think that "moonshine" in this case, at least, has produced a mental picture differing from that ordinarily observed after long continued overindulgence in alcohol.

DR. JULIUS GRINKER: I found the Achilles reflexes present in this patient, but no foot drop. Consequently, I disagree with the diagnosis of Korsakoff

psychosis due to alcoholism, but think the symptoms were due to indulgence in "moonshine."

DR. LEMCHEN: I have never known patients with Korsakoff's disease to recover within a day or two. Korsakoff described the syndrome in a country where they use a great deal more "moonshine" than we do here. Those patients seldom recovered entirely, as has this patient, without dementia and with good insight.

NEW YORK NEUROLOGICAL SOCIETY

The Four Hundred and Nineteenth Regular Meeting, March 3, 1925

I. ABRAHAMSON, M.D., *President, Presiding*

HUMAN EPIDEMIC ENCEPHALITIS IN RABBITS. DRS. M. NEUSTAEDTER, W. W. HALA and E. J. BANZHAF.

There is a wide divergence of opinion among investigators as to the possibility of successful transmission of human epidemic encephalitis to rabbits. Strauss and his associates Doerr and Zdansky, Levaditi and his associates McIntosh, Silberstein and others, report positive results.

On the other hand, Flexner and Amoss, Douglas and Boessermans and van Boeckel have failed to produce the disease in rabbits.

In September, 1924, we received from Doerr's laboratory in the University of Basel the brain of a rabbit that was supposed to be successfully infected with human epidemic encephalitis, showing the typical lesions and having been well controlled.

On Oct. 2, 1924, we began our experiments with the suspensions and filtrates of this rabbit brain, and of those rabbits that seemed successfully infected. Following is a summary of the results of our experiments:

Of five animals that were injected with suspension, three exhibited characteristic gross and microscopic pathology of encephalitis. One of these rabbits did not show a characteristic pathology. One died on the third day and exhibited a marked meningitis and hemorrhagic encephalitis.

Five rabbits received injections with respective filtrates of the various rabbits, and of these, four died, and one survived and was well after a month. Laboratory examination of all these rabbits did not reveal the characteristic perivascular round cell infiltration nor the neuronophagocytosis; but showed focal necrotic areas, perivascular and pericellular spaces dilated and hemorrhages, a reaction to a toxic state.

The antigen prepared from the filtrates gave good results, fixing complement in spinal fluids of patients that were acutely ill with epidemic encephalitis and poliomyelitis. All brain smears made at the time of necropsy showed no visible bacteria.

Cultures made from the brains in glucose broth and veal agar and incubated aerobically and anaerobically for one week remained sterile, with the exception of one contaminated with a hay bacillus. All sections whose pathology was characteristic of encephalitis were stained according to Ziehl-Neelsen's method, and nowhere could we find any *Encephalitizoa cuniculi.*

Conclusion.—We may therefore conclude that the encephalitis transmitted to the rabbits with the suspensions was produced by a specific virus; that this virus did not pass the Berkefeld filter; and that this virus could not be

obtained by ordinary aerobic and anaerobic cultural methods, and was not seen on the stained sections.

DISCUSSION

DR. J. H. GLOBUS: The problem is purely for a bacteriologist. I can only touch on a few points, particularly those which were brought to light by recent publications as well as by the earlier work on the subject of the transmission of the disease in question to experimental animals. The first experiments were attempted very early in the epidemic of so-called lethargic encephalitis by workers in one of the hospitals of New York City, who have reported the successful transmission of the disease. This work has been confirmed by other laboratories, but also met with a great deal of criticism from other quarters where similar experiments were carried out with negative results. The main objection to acceptance of the results of the first workers was based on the recent discovery that experimental animals (rabbits) often present symptoms and histologic changes typical of encephalitis, which, however, in rabbits are due to some protozoon body, encephalozoon, a protozoon disease common in rodents. Thus it is said that this condition when unrecognized may lead to wrong conclusions on transmissibility of the disease. It is rather significant that, in spite of this, these same critical investigators have themselves found it possible to obtain a virus and transmit it, so as to produce the disease picture in animals, and to carry this disease picture from one generation to another. Does it not in a way disqualify some of their objections? For the very same investigators who regard the protozoon infection as a stumbling block in the recognition of the pathologic picture in the experimental animal, characterize and describe their results as "typical histological" pictures in experimental animals produced by transmissible virus. At the same time, it is also claimed that the typical disease of acute epidemic encephalitis cannot be transmitted to experimental animals, since the clinical and pathologic picture in experimental animals does not correspond to that of human encephalitis. It is rather difficult to accept the criterion that in order to establish the possibility of transmission of a disease from the human organism to that of the experimental animal one must establish the same clinical picture. You cannot expect to produce in a rabbit a true parkinsonian picture, or some of the emotional display and facial expression seen in human acute encephalitis. Highly suggestive, nevertheless, is the fact that the same critical observers suggest that the virus of herpes labialis, which they think bears striking resemblance to the virus of experimental encephalitis, can be transmitted to the experimental animal and produce a picture which is said to be "typical" of acute epidemic encephalitis. It is true that there is a general feeling among neuropathologists that there is no such a thing as a "typical" histologic picture for acute epidemic encephalitis. As a matter of fact, there are several forms and varying degrees in the morphology of the disease. There is the common infiltrative type, passing into the subacute or chronic productive type in which the vessels are surrounded by scar tissue; and the fulminating serous or hemorrhagic type in which there are few or no infiltrations, but in which a great deal of destruction has taken place.

There is, however, one point which apparently has not been stressed enough, and that is that acute epidemic encephalitis is a disease which has, for some reason, a selective affinity for a certain part of the brain, for the midbrain. You are familiar with the typical picture as one which shows manifestations indicating involvement of the midbrain, the seat of the extrinsic and intrinsic eye mechanism as well as that of the coordinating centers.* This, to my

mind, is highly important, for it helps us to separate this disease from another form of inflammatory disease of the central nervous system, anterior poliomyelitis, which is regarded as being very close to the former in its etiology, though the causative factors have a selective affinity for other parts of the central nervous system. If we are to reproduce the disease experimentally, not only in the way of producing diffuse adventitial infiltrations or cloudy swelling of the cells, but also bringing about an inflammatory lesion limited to the midbrain, we could say that the disease is caused by a virus similar to that which causes acute epidemic encephalitis. So long as we cannot meet this condition, we cannot definitely state that the virus which produces a similar inflammatory lesion but which is indiscriminate in the selection of the parts of the brain is the same agent as that which causes acute epidemic encephalitis.

Dr. Bernard Sachs: The first point which seems to be of great importance is one to which Dr. Globus has referred. I would even be inclined to put it a little more strongly and to say that I do not think that any histologic picture can be presented as evidence of encephalitic disease transmitted from man to animal, or from animal to man. For, if you make a careful analysis of the histologic pictures that have been presented as characteristic of the experimental form of encephalitis or of poliomyelitis, you will find that these pictures are practically the same. The histologic picture seems to be evidence only of some infectious or inflammatory condition. It is evidence of infectious encephalitis, but it does not disclose the type of infection. Dr. Globus' point is well taken that if you could produce a midbrain encephalitis exclusively, take an emulsion of such a brain, and produce a similar lesion in the same region in another animal, you could then say that it was the lesion of encephalitis.

The rabbit seems to be a rather unfortunate animal to work with. According to statements made by Flexner and others, it is the subject of a good many diseases, particularly of the encephalitic type. Therefore, a rabbit after any sort of inoculation or injection is rather questionable evidence of the transmission of the disease from one animal to another, or from man to an animal. Flexner in one of his latest statements, in which he summarizes the subject in a very judicial way, has come to the conclusion that it requires further study and further discussion. We may therefore regard Dr. Neustaedter's paper as a contribution to a problem which still remains to be solved.

Dr. Neustaedter: I agree with every one who says that the pathologic picture alone is not typical of the disease. A slide showing perivascular infiltration and neuronophagocytosis indicates only an inflammatory reaction. That is also true of poliomyelitis. But we must remember that in poliomyelitis when we inject the animal intracerebrally, in the mucosa of the nose or intraspinally, we invariably get a characteristic lesion in the anterior horns and also in the brain, and the typical clinical picture. In 128 monkeys which I used in poliomyelitis with filtrates, in all cases a typical clinic picture and typical lesions were produced just as with the suspension.

I cannot accept Dr. Globus' contention that epidemic encephalitis is a midbrain disease exclusively, nor can I accept the statement that the only symptom we find is the parkinsonian syndrome. We find almost anything on the calendar. In more than 300 cases I have observed that, just as in neurosyphilis, you can find a clinical picture in accord with the site of the lesion. I do not want to give the impression that I believe that I have produced human epidemic encephalitis. If the virus is a filtrable virus, the same picture should

be produced with the filtrate as with the suspension, as I have always obtained in poliomyelitis. I was careful to state that I seem to have dealt with some specific virus that did not pass the filter and could not be cultured. The contention of all investigators who have produced the disease in the rabbit from saliva and from the nasopharyngeal discharges seems to be that it is a filtrable virus. It is not a protozoon infection. Another point is that I used four different lots of rabbits from the same dealer. I called to the attention of Rose that Flexner found in 50 per cent. of normal rabbits typical encephalitis lesions, but he claimed not to be able to find the disease in Switzerland in more than 2 per cent. Rosenow showed us his streptococcus infection producing symptoms in rabbits analogous to those of the patients, myoclonus, etc., and nobody else has done it. There is one point here which is of great interest to me—that the antigen of these filtrates has produced positive fixations. This work was very well controlled and done over and over again in cases of encephalitis. I have written Dr. Rose about it, sent him some slides, and explained the failure of the filtrate. He states that he had similar results, that he has not produced with the filtrate typical histologic pictures. When he has injected a suspension of 1:1,000 of these rabbits that did not show the characteristic picture, he has produced an immunity for three months against the virus of herpes, and for that reason he contends that it is epidemic encephalitis. The whole question is difficult, and I believe that all work on it is of value.

THE PUPIL IN SOMATIC AND VISCERAL DISORDERS IN ASSOCIATION WITH REFERRED PAIN AND HYPERALGESIA. DR. JOSEPH BYRNE.

After section or crushing of one sciatic nerve, the contralateral is the smaller pupil while the animal is quiescent, but it at once becomes the larger when the animal is disturbed or in pain. This is the pseudoparadoxic in contradistinction to the true paradoxic phenomenon. Both of these phenomena follow one-sided injury of the affective system; but the mechanism of the positive (dilatation) phase in each is different, the pseudoparadoxic phenomenon being fundamentally conditioned by hyperfunction of the injured primary affective neurons.

Unilateral experimental lesions below the level of the umbilicus induce the phenomenon in the contralateral eye, while unilateral lesions above that level induce it in the homolateral eye. In the abdominal viscera unilateral lesions with reference to the primitive mesenteric attachment evoke the phenomenon in the contralateral eye when the injured viscus receives its primitive afferent nerve supply through nerves that entered the spinal cord preponderately below the tenth or eleventh thoracic segment, and in the homolateral eye when the primitive afferent nerve supply preponderately entered the cord above that level. In accordance with this rule, lesions of the secreting portion of one kidney, ovary, or testis evoke the phenomenon in the homolateral eye, whereas unilateral lesions of the conducting system of each of these organs evoke it in the contralateral eye. Lesions of the liver, gallbladder and pancreas gave conflicting or negative results. In man, somatic and visceral lesions induce the pseudoparadoxic phenomenon in both of its phases exactly as they do in animals. Lesions in the following sites induce the phenomenon in the homolateral eye: (*a*) one lung, (*b*) either side of heart, (*c*) aorta (left eye), (*d*) stomach, duodenum, jejunum (one-sided lesion with reference to primitive mesenteric attachment), (*e*) spleen (left eye), (*f*) kidney cortex, ovary and testis. Unilateral lesions in the following sites induce the phenomenon in

the contralateral eye: (1) appendix caeci, colon, lower ileum; (2) renal pelvis, ureter and bladder; (3) vagina, uterus, one uterine tube; (4) epididymis, ductus deferens, seminal vesicles.

As it is the affective (pain) system of nerves that is concerned in the mechanism of the pseudoparadoxic phenomenon, the latter is naturally associated with conditions of pain and tenderness. This relationship has localizing diagnostic significance in visceral lesions when these evoke referred pain and more or less typical areas of hyperalgesia. The Head areas need revision and amending to be available in practice. In all painful lesions the phenomenon is under the examiner's control. Thus in a painful injury of the leg, the contralateral is the smaller pupil when the patient is quiescent, and not in pain, but it at once becomes the larger pupil when the patient moves or painful pressure is made on the injured part. As this reversibility is pathognomonic, we have in the pseudoparadoxic phenomenon an objective sign of pain which is useful not only clinically, but medicolegally, *e. g.*, in the detection of malingerers, etc. In visceral disease the phenomenon, especially when taken in cojunction with referred (reflected) pain and tenderness, supplies a valuable corroborative, localizing sign. The author has found it especially useful in differentiating lesions of the secreting system of the kidney, ovary and testicle from lesions of the duct systems of these organs. He has also found it useful in determining whether a renal calculus has passed and also in catheterization of the ureters.

DISCUSSION

Dr. Ernst Waldstein: Every one of us, whether general practitioner or nerve or eye specialist, owes a great debt of gratitude to Dr. Byrne for having devoted so many years of thoroughgoing research to the influence of the sympathetic system on the action of the pupils under pathologic conditions. Of course, most of us who preserve an interest in scientific research, in spite of the exigencies of daily practice, have been familiar with some outstanding facts relating to the connection which exists between the action of the pupils on one hand and our emotional life and certain pathologic phenomena on the other hand, such as affections localized in the neck, the thorax or below the diaphragm. Great progress has been made in this direction since the days when Claude Bernard made his first fundamental experiments. I dare say that hardly a week passes in an eye specialist's practice that he does not see one or two puzzling cases of inequality of pupils. In such cases, we were inclined to ascribe this phenomenon either to an unequal distribution of light entering the pupils or to the different angle at which it strikes the retina, bearing in mind the fact that the macular region of the latter reacts much more strongly than the more peripheral parts. There seems to be a difference also in the pupillokinetic energy of the temporal and nasal half of the retina, respectively, and possibly also of that part of the retina which is responsible for the so-called crescent in the field of vision, the last remnant in man in the phylogenetic evolution of the semidecussation of the optic fibers. Another point to be considered is the more or less known influence of the nervous vestibularis. Yet it is felt that only a part of such cases was sufficiently explained by the considerations mentioned above, and until recently sufficient material was not available to fill the gap. An entirely new light is thrown on many of these apparently misunderstood or half understood pupillar anomalies now that we are in a position to enjoy the fruits of the labor of such scientists as Dr. Byrne, and use the results of his work in our daily practice.

Mr. E. V. Broderick: Taking Dr. Byrne's work at its face value and assuming that everything stated is absolutely beyond contradiction, I believe that it is a contribution to be appreciated not only by medical men, but also by attorneys. Dr. Byrne has referred in passing to the fact that there are malingerers in our community. There can be no doubt about that. If there were an objective sign as regards pain which the medical man and the legal man could rely on as certain in determining whether there was existing pain, it would be a valuable contribution to all classes involved. Dr. Byrne's work indicates that the long sought for and much to be desired sign has been discovered.

On the question of time alone, if the medical man can be certain in diagnosis. and if the attorney can be satisfied that the medical man is right in his diagnosis, the contribution is valuable. Attorneys representing injured parties, and those representing the other party who has to pay out the money, are interested in the question of symptoms. I have no doubt that Dr. Byrne's work, in the field of law, as well as in medicine, has been directed to this question of pain, and I believe that the medical profession and the legal profession are both deeply indebted to him for his contribution. Hippocrates and Blackstone find common ground in his great accomplishment.

Dr. Riley: Will Dr. Byrne kindly explain the difference between the affective and the critical systems a little more clearly?

Dr. I Abrahamson: Have you tested a patient by approaching him with a sharp needle, not touching him, but seeing the effect on each pupil?

Dr. Byrne: As regards Dr. Riley's question, the affective system really means the pain system. Certain stimuli are used in the regular sensory tests, namely, superficial affective and superficial critical stimuli, and deep affective and deep critical stimuli. The superficial affective stimuli are pin-pricking, and heat and cold, *e. g.*, above 45 C. and below 25 C. For practical purposes 50 to 55 C. and ice are used in testing the affective (hurt) element in heat and cold. The superficial affective stimuli are therefore pin-pricking, and heat and cold of the degrees mentioned. Deep affective sensibility is tested by pressure — pain by 55 C. and ice in massive application. The superficial critical system is tested with the von Frey hairs (light touch) and with 37 to 39 C. (warmth), and 25 to 27 C. (critical element in cold). The so-called epicritic ranges of heat and cold described by Head are purely arbitrary. Heat is a complex of the simpler elements, warmth and hurt. The deep critical system is studied by testing posture and passive movement and by warmth, 37 to 39 C., and cold, 25 to 27 C., in massive application. In general, it may be said that the affective system mediates the qualitative element (hurt, pleasure, change of state) in any given stimulus and the critical system the quantitative element (postural and spatial recognition, etc.). The two systems are also differentiated by anatomic as well as by physiologic characteristics other than those mentioned.

In regard to Dr. Abrahamson's question, any stimulus, psychic or physical, which carries potential noxious value, will elicit the phenomenon.

Unusual Types of Spinal Cord Compression. Dr. Simon Rothenberg.

Dr. Rothenberg, in reporting four unusual cases of spinal cord compression spoke of the difficulties of diagnosis still extant despite present knowledge of physiology, localization and pathology of the cord lesions that has been enhanced through modern spinal surgery.

The first case was one of osteomyelitis of the spinal vertebrae with epidural abscess which caused complete compression of the spinal cord, without the usual accompanying symptoms of this disease, and simulated transverse myelitis. There was no rise in temperature until the course of the disease was far advanced, and the roentgen-ray and blood findings were negative.

The second case was that of spinal cord compression due to a *Cysticercus cellulosa,* the patient being a Jew and an observer of his faith. The symptoms in this case came on insidiously and gave no clue to the nature of the lesion.

The third case was that of a girl, aged 29, who had a syphilitic infection for which she had been treated for two years, but whose Wassermann reaction was still positive, and who had developed a spinal cord compression producing a partial paralysis of both lower extremities. She was finally diagnosed as having a cord tumor, and on operation an endothelioma was removed. The patient made a complete recovery.

The fourth case was that of a man, aged 24, who had suffered for nine years from Hodgkin's disease, and who suddenly developed a compression with flaccid paralysis of both lower extremities. He made a complete recovery after receiving one roentgen-ray treatment.

These unusual cases all seemed to show a rapid development of their symptoms shortly after the onset of the disease, making diagnosis of the respective conditions difficult.

SPINAL CORD TUMORS. MAX H. WEINBERG.

Three cases of cord tumors were presented showing unusual features, namely, the symptoms, diagnostic considerations, pathologic features, and therapy in case of an inoperable tumor.

The first case was one of hemangioma of the sixth thoracic vertebra in a young man aged 20. He became sick four months before operation. Both the sensory and motor symptoms were not constant for a while, and diagnosis was delayed until a permanent level was established. The tumor was not found at operation; but at necropsy the sixth vertebral body was found to be enlarged, exhibiting a protuberance which pressed on the cord. The eighth thoracic segment was compressed to one-half the size of the cord immediately below and above it. A lantern slide clearly demonstrated this feature. Another slide made from the tumor itself showed it to be composed of cavernous sinuses filled with blood, with but few spicules of bone present.

The second case was that of a cauda equina tumor in a woman aged 32. She had been sick for five months before admission. The most striking symptoms were severe pain in the lower part of the back on lying down, causing the patient to spend all her time in a wheel chair for the preceding five months; atrophy and flaccid paralysis of both legs and gluteal regions and bilateral toe drop. A distressing symptom was a sensation of extreme coldness of both lower extremities unrelieved by any measures taken to overcome it.

At operation, a large inoperable tumor of the cauda equina was found. Deep roentgen-ray therapy was resorted to, and the patient made a complete recovery. One and a half years after the operation she was found to be perfectly well and able to do the housework for a family of five.

The importance of deep roentgen-ray therapy in inoperable tumors was stressed, and similar reported cases were cited, especially from the German literature. The use of this treatment in all inoperable cord tumors was strongly urged.

The third case was that of a woman, aged 37, whose symptoms developed rapidly, within six weeks of admission, commencing with girdle pains which disappeared within four weeks. The paralysis came on rather quickly and was established in less than five weeks after the onset. She had a complete paraplegia, somewhat more marked on the left side. She had a modified Brown-Séquard syndrome in that the pain and temperature disturbances were more marked on the right side, and the paralysis more marked on the left. The level indicated an extradural tumor at the fourth thoracic segment. The diagnosis was rather difficult on account of the rapid development of the symptoms and the presence of a fairly high temperature, but all signs pointed toward a tumor of the fourth thoracic segment, and the patient was operated on.

The operative findings explained all the unusual features, as a tuberculoma of the dura was found. The tumor was shelled out easily, and sections showing typical tubercle formation were demonstrated. The patient is making a satisfactory recovery.

The literature dealing with this subject was reviewed, and the rarity of the condition was stressed. Only one similar case was referred to, that reported by Pelz.

Dr. Weinberg concluded: that cord tumors present many difficulties in diagnosis, necessitating careful study; that there are no absolute criteria to go by in diagnosing this condition; that exploratory laminectomies are justified in doubtful cases; and that deep roentgen-ray therapy is a valuable measure in inoperable tumors of the cord.

DISCUSSION

Dr. Byron Stookey: Dr. Rothenberg and Dr. Weinberg are fortunate in having so unusual a group of cases. I have never had the good fortune to encounter a cysticercus case. It is a very rare condition to find in this country, especially in an involvement of the spinal cord, with symptoms giving rise to those of spinal cord tumor. Endotheliomas in a patient showing a four plus Wassermann test are not so rare an occurrence. There is no reason why a patient with a four plus Wassermann reaction should not develop a neoplasm, and if after thorough antisyphilitic treatment no improvement has taken place, I see no reason why an exploratory laminectomy should not be performed provided the signs are sufficiently definite. At the Neurological Institute we have had very little experience with irradiation in spinal cord tumors, either by roentgen ray or by radium. I should think it would be of extreme value in the type of case to which Dr. Weinberg has called attention. It is extremely difficult in tumors of the cauda equina to remove the tumor in toto. While these are tumors within the vertebral canal, they are really peripheral nerve tumors, since they involve the peripheral nerves rather than the spinal cord itself. The prognosis, signs and symptoms are different from those in spinal cord compression. Cauda equina tumors have become extremely rare in our clinic. Dr. Elsberg in his earlier work saw quite a few, particularly giant tumors of the cauda equina, and was the first to describe these. However, within the last five years I do not recall seeing more than one such case.

I am very interested in the point to which Dr. Weinberg has called attention in one of his cases, namely, the increase of symptoms after lumbar puncture. I have seen several cases of intramedullary disease made worse by removal of the spinal fluid, and also one or two cases of suspected spinal cord tumor in which no tumor was found. I have no explanation to offer, but it is a fact which we must consider when we come to evaluate the importance of

the accentuation of symptoms after lumbar puncture in spinal cord tumors. However, it certainly remains a sign which should make us suspicious of the presence of compression of the spinal cord.

I think the very rapid onset of the case which Dr. Rothenberg reported certainly pointed to a myelitis, and I do not see how the diagnosis could have been made before. However, at the Neurological Institute we have had one or two patients with myelitis, with signs of complete obstruction, who have been operated on early, and who have walked out of the hospital. I recall two such cases; one a myelitis which came on after a curettage. This woman was a patient of Dr. Alfred S. Taylor. She was from Saranac, had tuberculosis, became pregnant and had an abortion. Following the abortion septicemia developed, and following the septicemia an acute transverse myelitis at the usual level, the thoracic ninth and tenth segments. Just why these segments are more frequently involved we do not know, but transverse myelitis frequently occurs at this level. Sir Victor Horsley called attention to the fact that these segments are more often involved in this type of lesion. This case showed a complete transverse myelitis. Dr. Taylor operated on the patient and she was discharged very much improved; she is now walking. I operated on another patient, a school teacher who had signs of acute transverse myelitis. Her illness began about the tenth day of her honeymoon. We exposed the cord, found it swollen and adherent to the dura. The patient was discharged from the hospital a month later. The third patient had a similar acute onset and was operated on; but she did not improve. I think, in view of the improvement in the other cases, that if they are seen early it is permissible to explore the cord.

I should like to call attention to the diagnostic procedure which has been used in the last few years more extensively than before, namely, the manometric study of the cerebrospinal fluid. No case of suspected spinal cord tumor can be considered as having been completely studied unless a manometric study has been made as a part of the neurologic examination. Certainly some of the cases which Dr. Rothenberg mentioned as being confused with spinal cord tumor could be ruled out by this procedure. In no case of multiple sclerosis have we ever found interruption of the cerebrospinal fluid circulation. This type of case, and also the so-called atrophic cord, which may give rise to neurologic signs so similar to spinal cord tumors, can be ruled out by a complete manometric study. It is a simple procedure. There are certain refinements which we have developed at the Neurological Institute which are perhaps helpful in determining an incomplete block. Every case of suspected spinal cord tumor must have a manometric study on the cerebrospinal fluid. We know that this water-bed is a very delicate system, so delicate that the lightest touch on even one jugular vein will produce a fluid wave and a rise in the manometer. This is a very delicate mechanism, and one which I think we shall be able to develop more as time goes on.

Obituary

ADOLF VON STRÜMPELL

1853-1925

Rarely do the work and the personality of a scientist merge as they did in Adolf von Strümpell, who died, Jan. 10, 1925.

We shall better understand Strümpell's character if we take note of some of the events in his early life. He was born, June 28, 1853, in Dorpat, Esthonia. As the son of a professor of philosophy, he acquired that clear and logical mode of thought that has since characterized his published works. I believe, too, that his tendency, even in studies that required concentration on the smaller details of disease, to keep the whole situation in mind and to endeavor to control the matter in hand instead of being controlled by it was a trait carried over from his youth.

In Leipzig, to which city his family had removed, he became the assistant of Wunderlich, a German clinician, to whom neurology owes an excellent early presentation of the subject. Erb also, though only a few years older than Strümpell,seems to have exerted a great influence over him. Strümpell was attracted by Erb's endeavors to free himself from the then dominant influence of the French in neurology and to replace diagnosis based on sensations by exact objective methods of examination.

Several important researches which constitute the basis for many conceptions that are still held date back to this early period in his career. For instance, who does not remember Strümpell's remarks "On Extensive Anesthesias and Their Effects on the Will, Body Movements and Consciousness," in which psychologic and clinical observations are merged together and on the basis of which he emphasizes in his "Contribution to the Physiology of Sleep" the significance of the elimination of receptive factors for the promotion of sleep?

In Leipzig it was doubtless Flechsig who awakened in the young scientist his interest in anatomy, while Weigert inspired in him a liking for the pathology of the nervous system. Thus in his first article "On Diffuse Cerebellar Sclerosis" and his "Studies on the Pathology of the Spinal Column," he took a definite stand on the questions of spastic paralysis of the spine and a combined systemic affection. Strümpell was the first to recognize the connection between spastic spinal paralysis, spinal muscular atrophy and amyotrophic lateral sclerosis; he was the first to describe the hereditary nature of this disease, separating it, at the same time, into an infantile and a juvenile group. In spite of all

this excellent early research, Strümpell would never have reached the position he attained in neurology if he had not written his famous textbook on specific pathology and therapy. It reveals all the excellent qualities characteristic of Strümpell's personality: a clear and logical arrangement of the material, a simple and convincing mode of presentation, uniform consideration of the pathologic and physiologic bases and of the clinical examination and an excellent simple yet forceful style, all of which contribute toward making it the standard work on clinical neurology. Strümpell was an excellent investigator, and he was the first to assign the proper significance to many symptoms; for example, the abdominal wall reflex in multiple sclerosis. He was also the first to explain the tibial phenomenon.

Soon after 1880, Strümpell had assumed a leading position in Leipzig. In 1883, he was called to the University of Erlangen as professor of internal medicine, where he remained until 1903. His studies on acute encephalitis in children were undertaken during the period at Erlangen. In this field he may be regarded as a predecessor of Ivar Wickmann, and at this time he formulated his theory concerning an inner relationship between poliomyelitis and acute poliencephalitis, and emphasized the infectious nature of both diseases. He was not so fortunate with respect to multiple sclerosis. Though he cleared up some of the clinical aspects of the disease, as regards the anatomic features, he retained the conception that multiple sclerosis was not an exogenous infectious disease but an endogenous affection, in which the primary manifestation was a hyperplasia of neuroglia and the secondary manifestation the destruction of the axis cylinder. Later, however, he accepted the view that there are exogenous types of the disease, which proves that he did not, like so many others, cling stubbornly to his first opinion, but allowed himself to be influenced by the advances in medical knowledge.

There are two diseases that bear Strümpell's name: the Strümpell-Marie type of spondylosis and the Westphal-Strümpell pseudosclerosis. We are indebted to Strümpell for a clearer conception of the general and the clinical aspects of spondylosis, though it is with regard to pseudosclerosis that his character is most clearly shown. He originated the idea of the amyostatic syndrome, distinguishing thus between myostatics and myodynamics. Whereas myodynamics is concerned with the movements of the muscles, myostatics has to do with all the forces that affect the bearing and posture of the body. We see here further evidence of Strümpell's endeavor through study of isolated phenomena to reach a fundamental conception under which single phenomena could be subordinated. In spite of many attempts to overthrow his conception, no one has been able to substitute anything better. It will be seen, further-

more, that this study of Strümpell's is characteristic of him in another direction. He does not overlook any symptom. He seeks to explain everything—to simplify whatever appears complicated. We are indebted to him, therefore, for the great progress that has been made in the study of that group of diseases that has been opened up to us by Wilson, Vogt, Hunt, and many other investigators. His article on this subject, as on many others, appeared in the *Deutsche Zeitschrift für Nervenheilkunde,* which through the efforts of Strümpell in collaboration with Schultze has become the leading organ of clinical neurology.

It is strangely pleasing that at a time when the Freud psychosis (for I know of no better word with which to designate the orgies of certain neurologists who make a special study of functional disorders) was at its height, Strümpell, as on many other occasions, struck the keynote when he said, "Neurosis cannot be an exclusively sexual problem." Freud's service to neurology does not lie so much in the better understanding of neurosis that he has given us as it does in the fact that he has made psychology of the individual a definite, inherent part of neurology. Strümpell combats the dogmatic aspects of Freud's views, but does not deny the great influence of sexual factors. How often I have heard him say: *"Das haben wir doch früher auch gewusst"* (that we knew before). Strümpell objected to the exaggeration of sexual factors so that they should come to be regarded as the alpha and omega of the hysteria problem. He held that there were essentially two types of hysteria, which every experienced neurologist will be compelled to recognize: a purely endogenous, constitutional type, and a type that develops on the basis of unsatisfied or suppressed desires—hysteria resulting from thwarted purposes. Strümpell does not attempt to deny the value and importance of psychanalysis. He simply cannot regard it as the beginning and the end of the neurosis problem. He considers it as one of the many methods by which the neurologist attains his goal.

Last year, Strümpell published a compendium to serve as an aid in examination of patients and in the diagnosis of the most important diseases. Although the compendium is short and much condensed, it contains the essentials in a precise, compact form, and reveals him as a master clinician.

I have not succeeded in even touching on all of Strümpell's activities. In 1903 he accepted a call to the University of Breslau, and in 1910 he removed to the University of Vienna, where he remained, only three semesters, however, so great was his longing to return to his beloved Leipzig, where he found everything that he demanded from life: scientific studies, service in the clinic, the companionship of congenial and eminent friends, and, above all, the many opportunities to hear the best music, which he enjoyed above everything else.

Strümpell was one of the best balanced individuals that I have ever known. He was a quiet, simple, good-natured man who furthered every scientific undertaking to the best of his ability. He was kind, forbearing and at times even indulgent to the students—ever ready to aid them in their work, and never assuming a superior demeanor. He was one of the world's great teachers of neurology because he possessed the widest experience and the most complete knowledge; because there was no field of his specialty with which he was not familiar, and, finally, because in every field he knew how to utilize his experience to the best advantage.

O. M.

Book Reviews

THE INHERITANCE OF MENTAL DISEASES. By ABRAHAM MYERSON. Cloth. Pp. 336. Price, $5. Baltimore: Williams & Wilkins Company, 1925.

Myerson reviews the vast literature on this subject, cites ninety-seven family histories from the records of the Taunton State Hospital, Mass., and details a number of experiments in heredity on animals, with the view of showing that psychiatrists in general have greatly exaggerated the rôle of heredity in mental disease, that clinical research in this field has been unusually biased (notably the work of Davenport and of Goddard), and that there is much evidence of the inheritance of acquired characters.

The book is divided into two parts. The first, largely polemic, is written with the lay reader in mind. Two chapters are devoted to the definition of common psychiatric terms and of the more frequent psychoses. Emphasis is placed on the nonhereditary factors in the production of feeblemindedness, epilepsy, the psychoneuroses and the psychoses, and the importance of regarding mental symptoms like any other disease phenomena. This is best illustrated in general paresis. At first only the mental signs were considered and classified under the purely social term "insane." Later, neurologic examinations and finally pathologic research revealed the "insanity" as one of the numerous manifestations of syphilis. The effects on the progeny, moreover, have no relation to the mental symptoms in the parent but are like syphilis in general. As in paresis in which only the biologic signs, not the mental, are constant, we are led to hope for similar discoveries in all the psychoses. Mental symptoms in the insane are regarded always as evidence of organic pathologic conditions as yet undiscovered in most cases. To talk, therefore, of the inheritance of mental disease is like talking about the inheritance of headache or any other symptom that may be related to numerous biologic units.

Nevertheless, in Part II, the major portion of the volume, containing statistical studies of 664 families in which there were 1,547 related cases of insanity, Myerson is compelled to work in the manner of his predecessors, utilizing the Kraepelinian classification of mental disease as so many inheritable units. The conclusions in the main are in agreement with the recent findings of other investigators with whose work comparisons are constantly made. From his own records Myerson concludes as follows: Insane mothers tend to have insane daughters more frequently than insane sons. In a large proportion of cases the insane descendants of insane become so at an earlier date than their ancestors (antedation). Four generations of mental disease in any one group is exceedingly rare. Persons with uncomplicated manic-depressive disease are followed in insane ancestry by descendants with manic-depressive psychosis, rarely also by feebleminded descendants. Those with dementia praecox also breed true. Usually the descendants have lower intelligence; often, however, they are feebleminded. The latter is regarded by Myerson in support of Kraepelin as dementia praecox with dementia as the prominent symptom. Paranoid condition in the ancestors breeds dementia praecox in the descendants, the psychosis usually being more severe and having an earlier onset in the latter. Involutional and senile states also, if paranoid, tend toward paranoid states and dementia praecox in the descendants; manic-depressive states of involution and senium tend toward manic-depressive

psychosis and dementia praecox, especially the latter. The psychoses of brothers and sisters tend on the whole to be alike, except that dementia praecox and feeblemindedness may occur in the one if the other has dementia praecox; but this may really be dementia praecox in which the dementia occurred earlier. It is unusual for dementia praecox and manic-depressive psychosis to occur in siblings. For the organic diseases and alcoholic psychoses there is no rule.

That the insane, feebleminded and epileptic tend to eliminate themselves through low birth rate is shown by the Taunton statistics as well as by Fernald and others. The marriage rate of patients with dementia praecox is one-fifth normal as compared with the general population; of alcoholic persons, two-thirds; of paretic patients and those with senile psychosis, the same. Moreover, the children of alcoholic, paretic, senile psychotic, epileptic, feebleminded and of schizophrenic persons are much fewer than those of normal persons.

The book ends with a review of experiments in animal heredity, including some of the author's own, showing a modification of germ-plasm in the descendants through poisoning the parent with chemicals (alcohol, lead), treating with roentgen ray, injecting lens-destroying serums and modifying the environment through temperature changes. From these data the author is strongly inclined to the belief that emotional damage may also affect the offspring through the germ-plasm, and that the life of the city especially is "blastophoric."

The style is direct, clear, personal and challenging. In spite of excessive citations of family histories and at times an unnecessarily caustic attitude, it is an interesting, thorough and scholarly presentation of a difficult subject.

YOUTH IN CONFLICT. By MIRIAM VAN WATERS, PH.D. Cloth. Price $1.00. Pp. 293. New York: Republic Publishing Co., 1925.

This book is a loose medley of observations and theories garnered from the author's experiences and philosophy and from reading on delinquency. Its central idea is the need and value of scientific work and community organization for the treatment of the young offender. Limited to a direct account of the author's long experience in this field, it would show more forcibly than it does the advantage of an enlightened procedure of juvenile court judges and probation officers over the barbaric adult-legal methods of handling "youth in conflict." The book stresses in graphic manner, and that is its chief merit, the fact that a criminal act is the result of a highly complex body of circumstances built up out of the particular social experiences, from earliest infancy on, of a given individual often influenced by physical and mental conditions requiring specially skilled examination. The training of juvenile court judges, probation officers and institutional workers should be in medicine and psychiatry rather than in law; their consultations with the physician and psychologist rather than with the courts.

The book opens with an enthusiastic foreword by Ethel S. Dummer unwittingly marred by grossly misleading statements honestly derived from the unwieldy literature on this subject. We learn (page vii) that "a girl of sixteen who had regressed to behavior of the second year is now doing well at school and in the new home found for her" — cited as an example of "cure" of a case of dementia praecox; that "when an emotional conflict is resolved, the intelligence quotient may run up thirty or forty points in three months"; that (p. x) "convulsions are psychogenic." Of these statements, the first is improbable, though possible; the second, if true, evidence of poor technic

in mental testing; the third, inaccurate. The progress of mental hygiene will not be furthered by overstatement and anecdote. The vivid sketches of the routine day in court, presented so admirably in chapter one, contain sufficient argument for the need of scientific procedure. The program outlined for the community in chapter twelve is well organized and presents a series of schemes already proved out in practice. Statistics of probation work show sufficiently well the value of that undertaking. There is little need therefore for extreme generalizations or for conclusions highly personal and easily contested.

That "practically no child ever brought before the juvenile court has a home that fulfilled the standards of the biologically healthy family group" is a vague generalization without defining such standards, and in view of the fact that many families with nondelinquent children present similar homes to those with delinquent ones, probably inaccurate. An interesting literary though scientifically unproved explanation for the truancy of an adolescent is cited by the author as follows: "The swift movement of his muscles, the change of scene, the excitement of the road at first gave him a feeling of relief and security and finally afforded him pleasure and satisfaction, and the habit of running away took possession of him body and soul." For the cure of a selfish girl, Betty, aged 16, the writer becomes rather punitive: "For girls like Betty a brief course of vigorous routine in a correctional school is the best treatment"; and the author is rather naive when she writes, "the teacher should have ability to detect early signs of emotional maladjustment in children"; and more so in the following: "gradually under guidance of the experienced social worker insight into motives comes almost without effort to the normal child."

We learn further in this book that little Elizabeth who was delinquent over a period of years and who had a mental disease, presumably manic-depressive psychosis, was cured by her aunt who refused to consider her abnormal; that "many nervous manifestations of adolescence yield to sheer growth"; that "newspaper selling is one of the largest contributing factors to delinquency among city boys."

The book is enlivened by the tremendous earnestness of the writer, her zeal and enthusiasm in the work. It is weakened by her constant willingness to swallow the literature without intellectual mastication.